Volume IV:
Cardiovascular and
Cerebrovascular
Disease
Part 1

## Report of the Secretary's Task Force on


U.S. Department of Health and

Human Services

Volume IV:
Cardiovascular and
Cerebrovascular
Disease
Part 1

## Report of the <br> Secretary's Task Force on

## Black \& Minority Health

U.S. Department of Health and

Human Services

## SECRETARY'S TASK FORCE ON BLACK AND MINORITY HEALTH

## MEMBERS

Thomas E. Malone, Ph.D., Chairperson
Katrina W. Johnson, Ph.D., Study Director

Wendy Baldwin, Ph.D
Betty Lou Dotson, J.D. Manning Feinleib, M.D., Dr.P.H. William T. Friedewald, M.D. Robert Graham, M.D.
M. Gene Handelsman Jane E. Henney, M.D. Donald R. Hopkins, M.D. Stephanie Lee-Miller

Jaime Manzano J. Michael McGinnis, M.D. Mark Novitch, M.D. Clarice D. Reid, M.D. Everett R. Rhoades, M.D. William A. Robinson, M.D., M.P.H. James L. Scott
Robert L. Trachtenberg
T. Franklin Williams, M.D.

## ALTERNATES

Shirley P. Bagley, M.S.
Claudia Baquet, M.D., M.P.H. Howard M. Bennett
Cheryl Damberg, M.P.H.
Mary Ann Danella, Ph.D.
Jacob Feldman, Ph.D.
Marilyn Gaston, M.D.
George Hardy, M.D.
John H. Kelso

James A. Kissko
Robert C. Kreuzburg, M.D.
Barbara J. Lake
Patricia L. Mackey, J.D.
Delores Parron, Ph.D.
Gerald H. Payne, M.D.
Caroline I. Reuter
Clay Simpson, Jr., Ph.D.
Ronald J. Wylie
Introduction to the Task Force Report. ..... v
Members of the Subcommittee on Cardiovascular and Cerebrovascular Diseases ..... ix
Report of the Subcommittee ..... 1
PART 2
Supporting Papers

1. Shiriki K. Kumanyika, Daniel D. Savage: Ischemic Heart Disease Risk Factors in Black Americans ..... 229
2. Lucile L. Adams, Laurence O. Watkins, Lewis H. Kuller, Daniel D. Savage, Richard Donahue, Ronald E. LaPorte: Relationship of Social Class to Coronary Disease Risk Factors in Blacks: Implications of Social Mobility for Risk Factor Change ..... 285
3. Hector F. Myers: Coronary Heart Disease in Black Populations: Current Research, Treatment, and Prevention Needs ..... 303
4. Melford J. Henderson, Daniel D. Savage: Prevalence and Incidence of Ischemic Heart Disease in United States' Black and White Populations ..... 347
5. Helen P. Hazuda: Differences in Socioeconomic Status and Acculturation among Mexican Americans and Risk of Cardiovascular Disease ..... 367
6. Shiriki K. Kumanyika, Daniel D. Savage: Ischemic Heart Disease Risk Factors in Hispanic Americans ..... 393
7. Shiriki K. Kumanyika, Daniel D. Savage: Ischemic Heart Disease Risk Factors in Asian/Pacific Islander Americans ..... 415
8. Shiriki K. Kumanyika, Daniel D. Savage: Ischemic Heart Disease Risk Factors in American Indians and Alaska Natives ..... 445
9. Lewis H. Kuller: Stroke Report ..... 477

## Background

The Task Force on Black and Minority Health was established by Secretary of Health and Human Services Margaret M. Heckler in response to the striking differences in health status between many minority populations in the United States and the nonminority population.

In January 1984, when Secretary Heckler released the annual report of the Nation's health, Health, United States, 1983, she noted that the health and longevity of all Americans have continued to improve, but the prospects for living full and healthy lives were not shared equally by many minority Americans. Mrs. Heckler called attention to the longstanding and persistent burden of death, disease, and disability experienced by those of Black, Hispanic, Native American, and Asian/Pacific Islander heritage in the United States. Among the most striking differentials are the gap of more than 5 years in life expectancy between Blacks and Whites and the infant mortality rate, which for Blacks has continued to be twice that of Whites. While the differences are particularly evident for Blacks, a group for whom information is most accurate, they are clear for Hispanics, Native Americans, and some groups of Asian/Pacific Is landers as well.

By creating a special Secretarial Task Force to investigate this grave health discrepancy and by establishing an Office of Minority Health to implement the recommendations of the Task Force, Secretary Heckler has taken significant measures toward developing a coordinated strategy to improve the health status of all minority groups.

Dr. Thomas E. Malone, Deputy Director of the National Institutes of Health, was appointed to head the Task Force and 18 senior DHHS executives whose programs affect minority health were selected to serve as primary members of the Task Force. While many DHHS programs significantly benefit minority groups, the formation of this Task Force was unique in that it was the first time that attention was given to an integrated, comprehensive study of minority health concerns.

## Charge

Secretary Heckler charged the Task Force with the following duties:

- Study the current health status of Blacks, Hispanics, Native Americans, and Asian/Pacific Islanders.
- Review their ability to gain access to and utilize the health care system.
- Assess factors contributing to the long-term disparities in health status between the minority and nonminority populations.
- Review existing DHHS research and service programs relative to minority health.
- Recommend strategies to redirect Federal resources and programs to narrow the health differences between minorities and nonminorities.
- Suggest strategies by which the public and private sectors can cooperate to bring about improvements in minority health.


## Approach

After initial review of national data, the Task Force adopted a study approach based on the statistical technique of "excess deaths" to define the differences in minority health in relation to nonminority health. This method dramatically demonstrated the number of deaths among minorities that would not have occurred had mortality rates for minorities equalled those of nonminorities. The analysis of excess deaths revealed that six specific health areas accounted for more than 80 percent of the higher annual proportion of minority deaths. These areas are:

- Cardiovascular and cerebrovascular diseases
- Cancer
- Chemical dependency
- Diabetes
- Homicide, suicide, and unintentional injuries
- Infant mortality and low birthweight.

Subcommittees were formed to explore why and to what extent these health differences occur and what DHHS can do to reduce the disparity. The subcommittees examined the most recent scientific data availahle in their specific areas and the physiological, cultural, and societal factors that might contribute to health problems in minority populations.

The Task Force also investigated a number of issues that cut across specific health problem areas yet influence the overall health status of minority groups. Among those reviewed were demographic and social characteristics of Blacks, Hispanics, Native Americans, and Asian/Pacific Islanders; minority needs in health information and education; access to health care services by minorities; and an assessment of health professionals available to minority populations. Special analyses of mortality and morbidity data relevant to minority health also were developed for the use of Task Force. Reports on these issues appear in Volume II.

## Resources

More than 40 scientific papers were commissioned to provide recent data and supplementary information to the Task Force and its subcommittees. Much material from the commissioned papers was incorporated into the subcommittee reports; others accompany the full text of the subcommittee reports.

An inventory of DHHS program efforts in minority health was compiled by the Task Force. It includes descriptions of health care, prevention, and research programs sponsored by DHHS that affect minority populations. This is the first such compilation demonstrating the extensive efforts oriented toward minority health within DHHS. An index listing agencies and program titles appears in Volume I. Volume VIII contains more detailed program descriptions as well as telephone numbers of the offices responsible for the administration of these programs.

To supplement its knowledge of minority health issues, the Task Force communicated with individuals and organizations outside the Federal system. Experts in special problem areas such as data analysis, nutrition, or intervention activities presented up-to-date information to the Task Force or the subcommittees. An Hispanic consultant group provided information on health issues affecting Hispanics. A summary of Hispanic health concerns appears in Volume VIII along with an annotated bibliography of selected Hispanic health issues. Papers developed by an Asian/Pacific Islander consultant group accompany the report of the Subcommittee on Data Development appearing in Volume II.

A nationwide survey of organizations and individuals concerned with minority health issues was conducted. The survey requested opinions about factors influencing health status of minorities, examples of successful programs and suggestions for ways DHHS might better address minority health needs. A summary of responses and a complete listing of the organizations participating in the survey is included in Volume VIII.

## Task Force Report

Volume I, the Executive Summary, includes recommendations for department-wide activities to improve minority health status. The recommendations emphasize activities through which DHHS might redirect its resources toward narrowing the disparity between minorities and nonminorities and suggest opportunities for cooperation with nonfederal structures to bring about improvements in minority health. Volume I also contains summaries of the information and data compiled by the Task Force to account for the health status disparity.

Volumes II through VIII contain the complete text of the reports prepared by subcommittees and working groups. They provide extensive background information and data analyses that support the findings and intervention strategies proposed by the subcommittees. The reports are excellent reviews of research and should be regarded as state-of-the-art knowledge on problem areas in minority health. Many of the papers commissioned by the Task Force subcommittees accompany the subcommittee report. They should be extremely useful to those who wish to become familiar in greater depth with selected aspects of the issues that the Task Force analyzed.

The full Task Force report consists of the following volumes:

Volume I: Executive Summary
Volume II: Crosscutting Issues in Minority Health:
Perspectives on National Health Data for Minorities Minority Access to Health Care Health Education and Information Minority and other Health Professionals Serving Minority Communities

Volume III: Cancer

Volume IV: Cardiovascular and Cerebrovascular Diseases

Volume V: Homicide, Suicide, and Unintentional Injuries
Volume VI: Infant Mortality and Low Birthweight

Volume VII: Chemical Dependency Diabetes

Volume VIII: Hispanic Health Issues
Survey of the Non-Federal Community Inventory of DHHS Program Efforts in Minority Health

CHAIR
William T. Friedewald, M.D.
Director
Division of Epidemiology and Clinical Applications National Heart, Lung, and Blood Institute National Institutes of Health

## MEMBERS

Howard Bennett, esq.
Associate Deputy Director Office of Management and Policy Office for Civil Rights

Allan L. Forbes, M.D.
Director
Office of Nutrition and Food Sciences
Center for Food Safety and Applied Nutrition
Food and Drug Administration
Mark Novitch, M.D.
Formerly: Deputy Commissioner
Food and Drug Administration
Paul A. Nutting, M.D.
Director
Office of Primary Care Studies Health Resources and Services Administration

Gerald H. Payne, M.D.
Chief
Prevention and Demonstration Research Branch
Division of Epidemiology and Clinical Applications
National Heart, Lung, \& Blood Institute
National Institutes of Health
Daniel D. Savage, M.D., Ph.D.
Medical Officer
National Center for Health Statistics
Ronald J. Wylie, esq.
Special Assistant to the Administrator
Health Care Financing Administration
T. Franklin Williams, M.D.

Director
National Institute on Aging
National Institutes of Health

Shirley P. Bagley, M.S.
Assistant Director for Special
Programs
National Institute on Aging
National Institutes of Health

Mary Ann Danello, Ph.D.
Special Assistant to the Commissioner
for Science
Food and Drug Administration

Lynn A. Larsen, Ph.D.
Associate Director for Program Development Center for Food Safety and Applied Nutrition Food and Drug Administration

## CONSULTANT WRITERS

Laurence O. Watkins, M.D., M.P.H.

Assistant Professor of Medicine Section of Cardiology
Department of Medicine Medical College of Georgia

Hector F. Myers, Ph.D. Associate Professor of Psychology<br>Department of Psychology<br>University of California at Los Angeles

## STAFF

Elisabeth Pitt, M.A.
Program Analyst
Clinical Applications and Prevention Program
Division of Epidemiology \& Clinical Applications
National Heart, Lung, \& Blood Institute
National Institutes of Health

Marilyn Kunzweiler, M.P.H.
Presidential Management Intern
Division of Epidemiology \& Clinical
Applications
National Heart, Lung, \& Blood Institute National Institutes of Health

# Report of the Subcommittee On Cardiovascular and Cerebrovascular Diseases In <br> Black and Minority <br> Populations 

All tables and figures from the American Heart Journal reprinted with permission by C.V. Mosby Company.

All material copyrighted by the American Heart Association reprinted by permission of the American Heart Association.

Reproduced with permission from the Annual Review of Public Health, vol. 2, © 1981, by Annual Rēviews, Inc.

Archives of Environmental Health, vol. 19, August 1969. Reprinted with permission of the Helen Dwight Reid Educational Foundation. Published by Heldref Publications, 4000 Albemarle Street, N.W., Washington, D.C. 20016 .

The table from Journal of Chronic Diseases, vol. 17, by Gordon T. GarciaPalmieri MR, Kagan A, et al.," Differences in coronary heart disease in Framingham, Honolulu, and Puerto Rico," 1974, reprinted with permission, Pergamon Press, Ltd.
ACKNOWLEDGEMENTS ..... xvii
INTRODUCTION ..... 1
CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN BLACK AMERICANS Introduction ..... 3
Coronary Heart Disease ..... 3
Mortality ..... 3
Morbidity ..... 5
Incidence ..... 5
Cohort Studies ..... 6
Sudden Death ..... 6
Acute Myocardial Infarction ..... 7
Hypertension ..... 7
Blood Pressure Levels and Prevalence of Hypertension ..... 7
Trends in Blood Pressure Levels and Prevalence ..... 9
Awareness of Blood Pressure Status, Treatment, \& Control ..... 10
Trends in Awareness, Treatment, and Control ..... 11
Stroke ..... 11
Mortality ..... 11
Morbidity ..... 13
Hypertensive End-Stage Renal Disease ..... 14
Introduction ..... 14
Morbidity ..... 14
Incidence ..... 14
Mortality ..... 15
Explanations for Differences ..... 15
Introduction ..... 15
Biologic and/or Physiologic Variables ..... 16
Coronary Heart Disease ..... 16
Introduction ..... 16
Age, Sex, and Family History ..... 17
Hypertension ..... 17
Blood Lipids and Lipoproteins ..... 19
Total Blood Cholesterol ..... 19
High-Density Lipoprotein Cholesterol ..... 19
Cigarette Smoking ..... 20
Diabetes Mellitus ..... 22
Electrocardiographic Abnormality ..... 23
Obesity ..... 24
Conclusion: Multiple Risk Factors ..... 25
Hypertension ..... 26
Introduction ..... 26
Inadequacy of Genetic Explanations ..... 27
BP Distributions in African \& Other Black Populations ..... 27
Differential Sensitivity to Dietary Electrolytes? ..... 28
Correlates of Hypertension Incidence: Implications ..... 29
for Primary Prevention
Stroke ..... 30
Hypertension ..... 30
Cholesterol and Cigarette Smoking ..... 30
Conclusion ..... 30
Hypertensive End-Stage Renal Disease ..... 30
Conclusion ..... 31
The Role of Hypertension in Black CVD Mortality and ..... 31Morbidity
Racial Trends in Hypertension-Related Mortality: Role ..... 31
of Hypertension Treatment
Socioeconomic Factors ..... 32
Social Epidemiology ..... 32
Coronary Heart Disease ..... 33
Social Epidemiology ..... 33
Socioeconomic Status and Biological Risk Factors ..... 34
Hypertension ..... 35
Social Epidemiology ..... 35
Psychosocial Stress Hypothesis ..... 36
Social Class and Hypertension-Related Mortality ..... 37
Stroke ..... 37
Links with Socioeconomic Factors ..... 37
Hypertensive End-Stage Renal Disease ..... 38
Links with Socioeconomic Factors ..... 38
Behavioral and/or Cultural Factors ..... 38
Introduction ..... 38
Coronary Heart Disease ..... 38
Health Beliefs/Knowledge ..... 38
Risk Factors ..... 39
Diet and Cardiovascular Disease ..... 39
Cardiovascular Health Education ..... 40
Health Practices ..... 40
Dietary Practices ..... 40
Physical Activity ..... 40
Cigarette Smoking ..... 41
Health Care Seeking Behavior ..... 42
Hypertension ..... 42
Health Beliefs/Knowledge ..... 42
Hypertension as a Health Problem ..... 42
Causes of Hypertension ..... 43
Health Practices ..... 43
Dietary Practices and Primary Prevention ..... 43
Health Care Seeking Behavior ..... 44
Nonadherence to Antihypertensive Therapy ..... 44
Determinants of Adherence ..... 45
Effective Intervention Models ..... 46
Access to and Utilization of the Health Care System ..... 47
Introduction ..... 47
Health Care for Coronary Heart Disease ..... 49
Data on Office Visits ..... 49
Hospitalization ..... 50
Prognosis After Myocardial Infarction ..... 50
Hospitalization for Chronic Coronary Heart Disease ..... 50
Coronary Arteriography and Coronary Bypass Surgery ..... 51
Health Care for Hypertension ..... 52
Data on Visits to Physicians ..... 52
Awareness, Treatment, and Control Status ..... 52
Perceptions of Access and Impact on Medical Care Use ..... 53
Effect of Decreasing Puilic Support for Health Services ..... 54
Black Physician Manpower ..... 54
CARDIOVASCULAR AND CEREBROVASCULAR DISEASE IN HISPANIC, ASIAN/PACIFIC ISLANDER, AND NATIVE AMERICANS
Introduction57
CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN HISPANIC AMERICANS Introduction ..... 59
Coronary Heart Disease ..... 60
Mortality ..... 60
Morbidity ..... 61
Stroke ..... 62
Mortality ..... 62
Hypertension ..... 62
Explanations for Differences ..... 64
Biologic and/or Physiologic Variables ..... 65
Risk Factors ..... 65
Lipids and Lipoproteins ..... 66
Hypertension ..... 67
Diabetes Mellitus ..... 67
Obesity ..... 68
Cigarette Smoking ..... 69
Socioeconomic and Sociocultural Factors ..... 70
Socioeconomic Factors ..... 70
Sociocultural Factors ..... 71
Behavioral and Sociocultural Factors ..... 71
Introduction ..... 71
Exercise ..... 72
Access to and Utilization of the Health Care System ..... 72
CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN ASIAN/PACIFIC ISLANDER AMERICANS
Introduction ..... 73
Coronary Heart Disease ..... 74
Mortality ..... 74
Morbidity ..... 77
Stroke ..... 77
Mortality ..... 77
Morbidity ..... 78
Explanations for Differences ..... 78
Biologic and/or Physiologic Variables in Japanese Americans ..... 79
Hypertension ..... 79
Cholesterol ..... 80
Smoking ..... 81
Overall Impact of Risk factors on CHD in Japanese Americans ..... 81
Biologic and/or Physiologic Variables in Chinese Americans ..... 82
Biologic and/or Physiologic Variables in Filipino Americans ..... 84
Hypertension ..... 84
Smoking ..... 84
CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN NATIVE AMERICANS Introduction ..... 87
Coronary Heart Disease ..... 88
Mortality ..... 88
Morbidity ..... 89
Explanations for Differences ..... 89
Biologic and/or Physiologic Variables ..... 90
Hypertension ..... 90
Blood Lipids and Lipoproteins ..... 91
Diabetes and Obesity ..... 92
Cigarette Smoking and Alcohol Use ..... 92
Socioeconomic and Sociocultural Factors ..... 92
TABLES AND FIGURES
Black Americans ..... 96
Hispanic Americans ..... 152
Asian/Pacific Islander Americans ..... 156
Native Americans ..... 186
RECOMMENDATIONS
Research Issues ..... 191
Information and Education ..... 197
Access and Utilization ..... 198
Capacity Building in the Non-Federal Sector ..... 199
Financing ..... 199
Health Professions' Development ..... 199
Leadership ..... 200
Data Issues ..... 200
REFERENCES
Black Americans (1. - 215.) ..... 203
Hispanic Americans (300. - 350.) ..... 219
Asian/Pacific Islander Americans (400. - 426.) ..... 223
Native Americans (500. - 524.) ..... 225

This report is the result of dedicated work done by the consultant writers, position paper authors, members of the Subcommittee, their alternates, National Heart, Lung, and Blood Institute staff, and the support staff from the DHHS Task Force on Black and Minority Health.

In particular, I would like to thank:
Laurence $O$. Watkins for his extensive and competent report on Black Americans

Hector $F$. Myers for his we11-crafted reports on Hispanic Americans, Asian/Pacific Islander Americans, and Native Americans, despite the scant data available to him;

Daniel D. Savage for his coordination efforts;
Elisabeth Pitt for her untiring, comprehensive, and competent editorial work in shepherding this report through its numerous stages, especially for her detailed work with its multiple authors;

Gerald H. Payne for his occasional representation of this Subcommittee at Task Force meetings, and for providing invaluable editorial advice.

This report drew heavily not only from the literature, but also from several manuscripts (position papers), commissioned by the Task Force for the Subcommittee, to the authors of which we are indebted. They are:

Lucile L. Adams, Laurence O. Watkins, Lewis H. Kuller, Daniel D. Savage, Richard Donahue, Ronald E. LaPorte

Helen P. Hazuda
Melford Henderson and Daniel D. Savage
Lewis H. Kuller
Shiriki K. Kumanyika and Daniel D. Savage
Hector F. Myers

In addition we wish to thank the following authors of papers, (commissioned for other Task Force Subcommittees) that this report used as resource materials:

John R. Davidson
E.S. Helena Yu, C.F. Chang, W.T. Liu, and S.H. Kan

Finally, gratitude is due to the many dedicated support staff on the Task Force who, despite other deadlines, managed to assist us in producing this report.

William T. Friedewald, M.D.
Director
Division of Epidemiology and Clinical Applications NHLBI, NIH

## INTRODUCTION

The arteriosclerotic diseases, particularly coronary heart disease and cerebrovascular disease, specifically stroke, cause more deaths, disability, and economic loss in the United States than any other group of acute or chronic diseases [Figure 1, Table 1]. They are also the leading cause of days lost from work. (1) Despite the fact that cardiovascular disease is a major killer in the United States, the data on the impact of cardiovascular diseases in minority populations are relatively sparse compared to those available on the white population. In recent years, the practice of collecting nationwide data by white and non-white categories is being replaced by data collection according to more specific minority categories. (2) In these minority groups, the most ample data on cardiovascular diseases are available for Blacks. The data on Hispanic populations, Asians, and Native Americans are less ample. Thus, though it is possible to examine some information on cardiovascular morbidity, mortality, prevalence and incidence rates, and their determinants in the white population, it is less feasible to obtain similar reliable data on cardiovascular diseases in specific minorities.

This report addresses the chronic cardiovascular diseases:

1. coronary heart disease, 2. hypertension, 3. stroke, and
2. end-stage renal disease related to hypertension (only for Blacks). The report consists of several parts, each devoted to a minority group: 1. Black Americans, 2. Hispanic Americans, 3. Asian/Pacific Islander Americans, and 4. Native Americans. The final parts consist of the recommendations of this subcommittee concerning cardiovascular and cerebrovascular health concerns in American minority populations, followed by the reference section.

## I INTRODUCTION

It is commonly recognized that hypertension is much more common in the Black than in the white population in the United States. Similarly, cerebrovascular disease (stroke) and chronic renal disease secondary to hypertension are known to be more common in Blacks than in whites. It is less commonly recognized that coronary heart disease may be as much a problem in Blacks as in the white population.(2) This review of cardiovascular disease will, therefore, consider first coronary heart disease, then hypertension and later the sequelae of hypertension, stroke, and hypertensive end-stage renal disease.

## Coronary Heart Disease

## (i) Mortality



* National data indicate that coronary disease mortality is sim- *
* ilar in Black and white men and greater in Black women than white*
* women. Although the data are inconclusive, some data on incidence*
* or the rate of development of new cases of CHD support the exis- *
* tence of a Black female excess. The currently available data *
$\therefore$ justify vigorous prevention and treatment efforts in Blacks. *


In a 1982 review, Gillum(2) summarized the available data on mortality and morbidity on coronary heart disease (CHD) in Black populations, with particular emphasis on the Black United States population. He noted that coronary heart disease is the leading cause of death among United States Blacks [Figure 2]. Recent national data indicate that, in comparison to white deaths, there is an excess of Black deaths attributed to coronary heart disease (ICDA codes $410-414$ ) in the age-group 20-64 years; the picture is reversed in those 65 years and older.(3) Data for 1979-1981 prepared for the Task Force by the National Center for Health Statistics(3) reveal that this is common to both Black men and women. Excess mortality from coronary disease is more marked in Black women than in Black men. Age- and sex-specific Black-white ratios of mortality rates have been computed from 1980 data [Table 2].(4) In the four decades of age from 25 to 64 years, the male Black-white ratios decrease progressively from 2.28 to 1.0 , while for women, the corresponding range is 3.30 to 1.78 . One implication of the data is that the CHD mortality rates for Black women are closer to those of Black men than is the case for whites. On the other hand, proportionate mortality statistics reveal that $17.5 \%$ and $20 \%$, respectively, of Black male and female deaths were attributed to coronary heart disease (CHD). The corresponding numbers for whites were $30.6 \%$ and $29.0 \%$ [Table 3].

Precise examination of trends in CHD mortality rates for Blacks is hampered by the fact that older data are reported only for the non-white category, without more specificity. In this category Blacks comprised more than $85 \%$ since 1940 . An analysis of these data by Gillum(2) reveals that between 1940 and 1967, CHD mortality rates rose sharply among non-white men and women, ages 35-74 years, so that by 1948 the age-adjusted CHD mortality rates in non-white women exceeded those of white women and the non-white to white mortality ratio increased steadily until 1968 . For men, non-white rates increased steadily to exceed those of white men by 1968, and have remained similar since then [Figure 3].

Since the mid 1960s, the age-adjusted CHD mortality rate for the entire United States population has declined.(5) The existence of such a decrease has been confirmed through 1982.(6) The decline actually began in the 1940s among white women, but not until 1969 among Black women. (7) Among white men, CHD mortality rates in successive cohorts have decreased since 1965. In contrast, CHD mortality rates for non-white men did not begin to decline until 1969. Between 1968 and 1976, age-adjusted CHD mortality declined in all groups except white women by $60-70 / 100,000$. The decline in white women was $34 / 100,000$. The percentage decline for non-white men was 29.2 and for non-white women 34.9; these exceeded the declines for white men and women respectively, 18.6 and 23.6 . The decline in deaths attributable to acute myocardial infarction in the same period for non-white men was $27 \%$ and non-white women $30 \%$. For whites, the corresponding rates were similar. In contrast, chronic CHD (ICD 412 ) caused $15 \%$ and $26 \%$ fewer deaths in non-white men and women, and the decrease in white men and white women was somewhat less, $6 \%$ and $16 \%$.

Gillum $(2,7)$ has suggested that examination of mortality rates is complicated by the following problems:

1. The past failure of the National Center for Health Statistics to report mortality for Blacks and other non-white groups separately before 1979.
2. Different population-age structures and CHD mortality color ratios at different ages make published generalizations of Black-white trends using age-adjusted rates misleading.
3. Death certificate diagnoses are likely to be more inaccurate in Blacks than whites.
4. The effects of changes in disease classification on Black mortality rates are inadequately documented.
5. Influenza epidemics have had a greater impact on CHD mortality in Blacks than in whites.
6. Census data for Blacks are likely to be more inaccurate than those for whites.
Gillum(7) has concluded that, though these problems undermine confidence in the accuracy of CHD mortality rates for Blacks, especially before 1967, "the data are certainly accurate enough to support the conclusion that coronary heart disease is the leading cause of death among U.S. Blacks." [Table 4]

* 

$\therefore$ The prevalence of coronary heart disease in Rlacks and whites *

* appears to be similar. Sampling techniques in national surveys *
$\therefore$ have not allowed definitive resolution of the question of dif- *
二
ferences in prevalence.
* 

*-

Methodologic problems make comparisons of CHD prevalence in Blacks and whites less revealing than might be expected. (8) In the 1960-62 National Health Examination Survey, (9) the prevalence of definite or suspect CHD, diagnosed by electrocardiographic (ECG) evidence of healed myocardial infarction (MI) or myocardial ischemia, or a history of MI or angina pectoris, was $4-6 \%$ in all race-sex groups [Tables 5-6]. The prevalence of ECG evidence of MI in adults, ages 18 to 79 years, was less than $2 \%$ within the "definite" category, 2\% of white men had ECG evidence of MI, compared to $1.7 \%$ of Black men. In women, the corresponding rates were $0.7 \%$ and $0.4 \%$. Blacks constituted $10.5 \%$ of this representative sample. In the 1971-75 National Health and Nutrition Examination Survey (NHANES I), (10) ECG evidence of healed MI was more common in white men than in Black men only above the age of 65 years, but was more common in Black women than in white women of similar age [Table 7]. If significant, these differences might reflect the higher age-specific CHD mortality in non-white men than in white men in the decades of age 35-44, 45-54, and 55-64 years, (11) so that the lower frequency in older Black men might reflect the fact that these men have survived the period that is for their group the one of highest mortality risk.

In the hypertensive stepped care subjects in the Hypertension Detection and Follow-up Program, (12) ECG-MI was detected at baseline in less than $2 \%$ of all race-sex groups, and a history of myocardial infarction diagnosed by a physician was given by about $5 \%$ of subjects in each group. The similar prevalence rates of MI in hypertensive subjects of both races were associated with similar all-cause mortality rates in the subsequent five years in each race-sex group. However, the data suggest that a Rose Questionnaire diagnosis of angina pectoris is less specific for CHD in Black women than in white women, since the five-year mortality rate in Black women with angina pectoris was much lower than that in similar white women.

## (iii) Incidence

[^0]The data on the incidence of CHD in Black U.S. populations are inadequate. This is because relatively few studies that included significant numbers of Blacks have been performed.

The studies of CHD incidence that examined biracial populations have been performed mostly in the rural south, and it is unclear whether the results are applicable to the entire U.S. Black
population. Over seven and one quarter years of follow-up in Evans County, Georgia from 1960-1967, the age-adjusted incidence of all manifestations of CHD was $24 / 1000$ in Black men and 83/1000 in white men, which yields a Black-white relative risk of .29.(13) (These short-term results were inconsistent with the observations on 20-year CHD mortality in this cohort. Black and white rates were similar.(14)) There was no significant difference in seven-and-one-quarter-year CHD incidence between Black and white women ( 34 vs $38 / 1000$ ). In the 14 -year follow-up (1960-62 to 1974-75) in the Charleston Heart Study in Charleston County, South Carolina, age-adjusted CHD incidence rates for random samples of Black and white men were 132 and 188/1000.(15) The picture was reversed for women: the rates for Black women exceeded those of white women, 161 vs $114 / 1000$. A small cohort of high social status Black men had the lowest rates, 61/1000 [Figure 4].

## (b) Sudden Death

The incidence of sudden cardiac death attributable to CHD has been examined, but no firm conclusion is possible. A 1964 New Orleans study revealed a rate of sudden death (within one hour of onset of symptoms) five times as high in Black men, ages $35-44$ years, as in white men, and in those aged 45-64 almost one-and-a-half times as high. ( 16,17 ) In Baltimore in 1964-65,(18) the sudden death rate in Black women was almost twice as high as in white women, whereas the rate in Black men was $62 \%$ of that observed in white men. Data from Nashville, Tennessee, 1967-68,(19) indicate slightly higher rates of death within 24 hours of onset of symptoms in Black men than in white men, and rates in Black women twice those of white women [Figure 5]. In both sexes, the proportions of subjects who died suddenly compared to those who were admitted to the hospital with MI, were higher in Blacks than in whites. In a 1970-72 Baltimore Sudden Death Study(20), no racial differences in the onset of sudden CHD death within 24 hours of the onset of symptoms were observed. In the Charleston Heart Study(15), the 14 -year age-adjusted incidence of sudden death (1960-74) was significantly higher in Black men than in white men (rate ratio 3.2). In Black women, though the rate was higher (rate ratio 1.6 ), the difference was not statistically significant. Thus, some data indicate similarity of sudden death rates for Blacks and whites, though in some locations there is evidence of a Black male excess, and in others of a Black female excess rate.

A number of studies have examined the frequency of hospital admissions for acute myocardial infarction (MI) in geographic areas with defined populations. (19,21-24) These data have been summarized by Henderson and Savage. (10) In some of these studies $(19,21,23,24)$ there has been a higher incidence of acute MI in white men than Black men, but some data indicate that the proportion of Black men with an MI admitted to hospital might have been lower than in whites, and the proportion of out-of-hospital deaths higher. (19,20,22) In addition, age-specific comparisons (by decades of age) of acute MI incidence have yielded conflicting results. In Newark, NJ, in 1973, Black male and white male rates were similar in the four decades of age 40-79 years(22), whereas in Nashville, TN, in 1967-68, there was a substantial white excess in men 55-74 years.(19)

Rates of hospital discharges for acute myocardial infarction in the National Hospital Discharge Survey, $1981(8,25)$, are more than twice as high for white men as for Black men 45-64, 65-74, and 75+ years old. Only for 25-44 year-old men are the rates similar. The Black female rates exceed those of whites at ages $25-44$ years, are similar in the 65-74 year group, and are substantially less in the 45-64 and 75+ years groups [Table 8]. These statistics are based on the first-listed diagnosis in the hospital discharge record and include those discharged both alive and dead. However, the diagnoses have not been verified by examination of clinical data, and no distinction could be made between initial and recurrent infarction. Surveillance of CHD events in biracial communities is currently being performed under the sponsorship of the National Heart, Lung, and Blood Institute (NHLBI), and should clarify this issue.

## Hypertension

## (i) Blood Pressure Levels and Prevalence of Hypertension


#### Abstract

 * Mean blood pressures are greater in Blacks than in whites. There * * is an excess of definite hypertension, borderline hypertension, $\dot{\star}$ * and isolated systolic hypertension in Blacks compared to whites. *为为


Hypertension or substantially elevated blood pressure is a chronic condition that increases the risk of circulatory diseases, particularly heart disease and stroke. There is a high prevalence of hypertension in the U.S. Black population; the most recent national data show that among adults, ages $18-74$ years, the prevalence of definite hypertension in Blacks is 1.4 times that observed in the white population.(26)

This Black excess of hypertension has been known for almost three decades. It was first confirmed in an epidemiologic survey in a defined biracial population in 1954. (27) In this study in Muscogee County, Georgia, Comstock observed that mean systolic and diastolic
blood pressures were higher in Blacks than whites, both male and female, and that there was a general tendency for all mean blood pressures to increase with age. Since then, a number of nationwide surveys have confirmed this result. The most recent data are those obtained in the National Health and Nutrition Survey of 1976-80 (NHANES II).(26) Earlier surveys include the National Health Examination Survey of 1960-62 (NHES I)(28), and NHANES I (1971-75). $(29,30)$ A forthcoming publication from the National Center for Health Statistics examines blood pressure levels and trends from 1960 to 1980 in people 18 to 74 years of age. (31)

The NHANES II data $(26,31)$ show that at ages $18-24$ and 25-34 years, mean systolic blood pressure levels for Black men were less than those for white men, though the differences were not statistically cignificant. However, in the age-groups 35-44, 45-54 and 55-64 years, mean systolic blood pressure levels were significantly greater in Black men than in white men. Similarly, among women in the age range $35-74$ years, systolic blood pressure levels in Black women were higher than those in white women. The same pattern was observed for diastolic blood pressure among both Black men and women at ages $35-74$ years. NHANES I reported regional variation of blood pressure levels. Mean systolic blood pressure levels were $8-9 \mathrm{~mm} \mathrm{Hg}$ higher in Black women in the South than in the Midwest and Northeast. (30) No such variation was observed in Black men. No consistent or significant regional pattern was observed for diastolic blood pressure.

Elevated blood pressure levels, defined as systolic blood pressure of at least 160 mm Hg or diastolic pressure of at least 95 mm Hg or both, was detected in $14.5 \%$ of all adults, Black and white, ages 25-74 years. The prevalence increased from $5.5 \%$ in the 25-34-year range to $26.6 \%$ at ages 65-74 years. In the 25-74-year range, elevated levels were significantly more prevalent among Black than white adults, $22.8 \%$ compared to $13.5 \%$. The rates among Black men were not significantly higher than those among Black women.

Definite hypertension, defined as blood pressures in excess of the critical levels given above and/or by the use of antihypertensive medication, was present in $22 \%$ of adults ages $25-74$ years. There was an excess of definite hypertension in Black adults. The prevalence in Black men (28\%) exceeded that in white men (21.2\%); that in Black women (39.8\%) exceeded that in white women (20.0\%). The Black excess is much more marked among women than men and more Black women are being treated than men. Mean systolic and diastolic blood pressure levels were noted to be inversely related to the amount of formal schooling received by examinees in all race and sex groups. For adults ages $18-74$ years (a less restricted range than discussed above), the prevalence rate was $17.7 \%$. The overall prevalence among white adults was $16.8 \%$ : $17.1 \%$ in white men and $16.6 \%$ in white women. The overall prevalence rate among Black adults was $25.7 \%$, this being significantly greater than that for white adults. The overall prevalence rate for Black women was significantly greater than that for Black men, $29.5 \%$ vs $21.1 \%$. Black women above the age of 45 years were significantly more likely to be classified as having definite hypertension than Black men in the same age range. The
prevalence rate for definite hypertension among Black women, ages 65-74 years, was the highest age-race-sex-specific rate detected: $60.1 \%$.

In addition to the 25.1 million adults ages $18-74$ years, with definite hypertension, there were 17.1 million adults of that age with borderline hypertension, defined as systolic blood pressure greater than or equal to 140 mm Hg and less than 160 mm Hg and/or diastolic blood pressure greater than or equal to 90 mm Hg and less than 95 mm Hg in subjects not taking antihypertensive medication. The prevalence is $11.9 \%$ for white adults and $12.5 \%$ for Black adults.

Isolated systolic hypertension, defined as the presence of a systolic blood pressure greater than or equal to 160 mm Hg but a diastolic pressure of less than 90 mm Hg , was more prevalent in Black adults, ages 55-74 years, (8.1\%) than among white adults of similar age (4.8\%).

## (ii) Trends in Blood Pressure Levels and Prevalence

\%
$\%$ In the period 1960-1980, mean systolic blood pressure has dec- $\quad *$

* lined more in Blacks than in whites. The prevalence of definite *
* hypertension increased significantly in whites, but did not *
* change significantly in Blacks. *

It is possible to assess differences in mean blood pressures across surveys, and such an analysis has been performed for the first systolic blood pressure measurement in NHES I, NHANES I and NHANES II. (31) This analysis reveals that mean systolic blood pressure has decreased significantly in the 20 -year period between 1960 and 1980 , in Black men from 138 to 136 to 130 mm Hg , and in Black women from 138 to 135 to 126 mm Hg . The age-adjusted mean systolic blood pressure declined more in Black adults than in white adults, 8 mm Hg compared to 4 mm Hg in men, and 12 mm Hg compared to 6 mm Hg in women. The improvement was greater for the older age-groups than for the younger.

Over the 20 -year period, the age-adjusted proportions of people with systolic blood pressure greater than or equal to 140 mm Hg decreased substantially more in Black adults than in white adults. From 1960 to 1980 , the decline for Black men was from $41.2 \%$ to $28.3 \%$, and for white men $32.4 \%$ to $27.0 \%$. For Black women the decline was from $39.6 \%$ to $26.7 \%$; for white women from $26.1 \%$ to $21.1 \%$. The net result is that these proportions are similar in three of the four race-sex groups in the 1976-1980 NHANES II data, $27.0 \%, 28.3 \%$ and $26.7 \%$ for white men, and Black men and women, respectively, with white women constituting $21.1 \%$.

The available data also allow comparisons of prevalence rates of hypertension in 1960-62, 1971-75, and 1976-80.(31) [Table 1] Freeman et al(32) have demonstrated, using appropriate statistical methods, that there were no significant changes in prevalence in age-race-sex-specific groups between the National Health Examination

Survey I(1960-62) and NHANES I (1970-74). The comparison of overall prevalence between NHES I and NHANES II reveals that in adults, ages 18-74 years, the proportion of the population with definite hypertension increased significantly for whites from $18 \%$ in 1960-62 to $21 \%$ in 1976-1980. Among Black adults, a nonsignificant decline was observed, from $33.6 \%$ in 1960-62 to $28.6 \%$ in 1976-80.

## (iii) Awareness of Blood Pressure Status, Treatment, \& Control



* By 1971-1975, Black adults were more likely to be aware of their * * elevated blood pressure status than whites. Hypertensive Black *
* adults were as likely as whites of the same sex to be treated * * with antihypertensive medications and to have their blood pres- * * sure controlled. $*$

NHANES II(26) also yielded information on awareness by hypertensive subjects of their blood pressure status, on medication usage by hypertensive subjects, and on the adequacy of blood pressure control in treated hypertensive subjects. Race-age-sex-specific rates are available for adults, ages 25-74 years, for hypertension awareness, treatment, and control [Table 9]. Overall, $26.6 \%$ of adults, ages 25-74 years, reported that they had never been told by a doctor that they had high blood pressure or hypertension. This was less likely among Black men than white men ( $35.7 \%$ vs $40.6 \%$ ), and among Black women than white women ( $14.5 \%$ vs $25.2 \%$ ), the difference being statistically significant only for women. In particular, for Black women ages 55-74 years, the proportions with undiagnosed hypertension were lower than for white women in the same age-group. However, Black men are less likely to be aware of having hypertension than Black women.

Medication use among hypertensives was similar in race-sex-specific groups. In fact, $38.3 \%$ and $40.9 \%$ of white and Black male hypertensives, respectively, reported being treated with medications. The corresponding numbers for women were $58.6 \%$ and $60.6 \%$. None of the within-gender differences was statistically significant. However, Black women with hypertension are more likely to be treated than Black hypertensive men. There were no significant differences between race-sex-specific rates for hypertension control by medication. For white and Black men, the rates were $20.9 \%$ and $16.1 \%$, and for white and Black women $40.3 \%$ and $38.3 \%$. The differences between men and women in both races were statistically significant.

$\therefore$ Though there was a significant excess of hypertension in Black $\div$ $\%$ men and women in 1976-1980, the trends in awareness, treatment, * * and control during the 1960-1980 period indicate significant im- * * provements in the status of Black adults.

The NCHS has published an examination of trends in awareness, treatment, and control status from 1960-1980. Unlike the NHANES II data on awareness, treatment, and control reported in earlier sections, these data are based on the population, ages 18-74 years, and report the frequency of awareness, treatment, and control in this population, rather than as a proportion of hypertensive subjects. This is appropriate since the prevalence of hypertension has increased significantly, at least in whites, from 1976 to 1980.

The data reveal substantial improvements in all categories for both racial groups. The proportion of people with undiagnosed hypertension has declined from 1960-1980.(31) Over the 20-year period, the age-adjusted proportion of people, ages $18-74$ years, with undiagnosed hypertension decreased from $10.7 \%$ to $9.8 \%$ in white men, from $7.7 \%$ to $5.6 \%$ in white women, from $21.1 \%$ to $9.9 \%$ in Black men, and from $13.2 \%$ to $4.5 \%$ in Black women. This decline was substantially larger in Black than in white adults.

The age-adjusted proportion of people, ages 18-74 years, taking antihypertensive medication increased from $3.8 \%$ to $7.6 \%$ for white men, from $7.0 \%$ to $11.1 \%$ for white women, from $6.0 \%$ to $9.2 \%$ for Black men and from $15.9 \%$ to $19.3 \%$ for Black women. Again, the age-adjusted proportion of people taking medications was generally higher among Black adults than among white adults, and among women than men, but the amount of increase from 1960-1980 was similar for each of the four race-gender groups.

The proportion of people, ages 18-74 years, with controlled hypertension increased significantly from 1960-1980 and was at least doubled for each race-gender group: from $1.5 \%$ to $3.4 \%$ for white men, from $2.9 \%$ to $6.3 \%$ for white women, from $0.6 \%$ to $3.3 \%$ for Black men, and from $6.0 \%$ to $11.6 \%$ for Black women.

## Stroke

## (i) Mortality

| ```* Stroke death rates are much higher in Blacks than in whites. * Death rates from stroke have been decreasing since the 1930s and * in recent years the rate of decline has been greater in non- * whites than in whites, especially in younger Blacks.``` |
| :---: |
|  |  |
|  |  |
|  |  |
|  |  |
|  |  |

Stroke is another cause of the long-standing excess of deaths among Blacks compared to whites. In 1969-71, stroke mortality rates for Black males exceeded those of white males by $44.3 \%$; the female excess was $46.0 \%$. This Black-white differential still persists, though it is noteworthy that the proportion of excess deaths has decreased in Black women in the ensuing 10 years.

In 1981 there were approximately 163,000 stroke deaths, $5.4 \%$ of which were among Black men and $6.5 \%$ among Black women [Table 10].(33) Age-adjusted stroke death rates are approximately twice as high among Black men and women compared to white men and women (rate ratios 1.9 and 1.8 , respectively). The existence of an excess of stroke deaths can also be inferred from the proportion of deaths due to stroke among Blacks: 7.2\% of deaths among Black men and 9.7\% among Black women were due to strokes in 1981, compared to $5.3 \%$ among white men and $8.2 \%$ among white women. The Black-white ratios of stroke mortality in specific age-sex groups (1980 data) indicate the disproportionate extent to which younger Blacks are at risk [Table 11]. In each decade of age from 25-34 years through 55-64 years, the Black/white ratio of stroke mortality among men exceeds three. It is as high as 4.5 in the 35-44-year age-group. Similarly, among women the ratio declines slowly from 3.5 in the age-group 25-34 years to 2.8 in the age-group 55-64 years. Thus, Black adults in these four decades of adulthood are at a marked excess risk of stroke mortality.

Data from a recent, biracial, male, cohort study confirm these observations concerning the Black excess. (34) During five-year follow-up of the 23,490 Black men and 325,384 white men who underwent screening in 18 U.S. cities in 1973-75 for the Multiple Risk Factor Intervention Trial, the cerebrovascular disease mortality rate was 2.6 times as high among Black men as among white men, 1.3/1000 compared to $0.5 / 1000$.

The available data reveal substantial variation in stroke mortality by geographic area; this is an observation of long-standing [Table 12].(33) These regional differences may be related in part to regional variation in systolic blood pressure levels, at least in women. In 1969-1971, when Black men, ages 45-54 years, in Savannah, Georgia were compared to white men of similar age in Baltimore, Maryland(35), the Black-white stroke mortality rate ratio was 10 . In 1978, the ratio was 4. A similar 1978 comparison of women revealed a ratio of 3.5 . In general, the highest rates have been detected among Black men in the Southeast, which has led to this area being described as the "Stroke Belt".

Stroke deaths have declined in the United States since the 1930s and this has been observed in both Blacks and whites [Table 13]. $(36,36 \mathrm{~A})$ The rate of decline in stroke mortality has increased in recent years. The percentage decline between 1968 and 1978 was $40.1 \%$. The decline has been greater in non-white than in white people, and has been greatest in non-white women, $49.1 \%$ compared to $38 \%$ in white women. The decline in stroke mortality has been observed in both the high and low stroke death rate areas in the United States [Table 14]. It is noteworthy that among Black women the rate of decline in the
period 1970-1980 has been greatest in the 35-44 year-old group, the group which manifested the highest disparity with white rates in earlier years [Table 15]. Among Black men, the greatest declines have been observed in the two decades of age: $35-44$ and 55-64 years.
(ii) Morbidity

为

* A higher proportion of Blacks than of whites in the population *
* has suffered stroke. $\quad$ *


Data on stroke morbidity in minorities are less ample. The 1972 National Health Interview Survey (37) yielded an estimated prevalence of stroke among whites of $7.2 / 1000$ and among non-whites 9.1/1000. Among the participants who were randomly assigned to the Hypertension Detection and Follow-up Program in 1973-1974(38), the prevalence of stroke was twice as high among Black men as among white men, while for women the Black-white ratio was 1.4. In 1967-69, there was a much more marked disparity in the prevalence of stroke between the Black and white racial groups in Evans County, Georgia (an area within the stroke belt).(39) The age-adjusted prevalence of stroke in Black women'was $43.4 / 1000$ compared to $15.0 / 1000$ in white women, while there was less of a disparity among men, the rates being 58.6/1000 among Black men and 53.2/1000 among white men.

The incidence of stroke is higher among Blacks than among whites. The age-adjusted incidence of stroke in southern Alabama in 1980 was $208 / 100,000$ among Blacks and $109 / 100,000$ among whites. (40) The highest age-adjusted rate was observed among Black women, $236 / 100,000$, compared to $88 / 100,000$ among white women (relative risk, 2.7), while in men the comparable rates were $172 / 100,000$ for Blacks and $139 / 100,000$ for whites (relative risk, 1.2). Similarly, the incidence of stroke was much higher among Black men and women in the Hypertension Detection and Follow-up Program than in white adults.(38)

Clinical evaluation of the type of stroke in the southern Alabama study revealed that the majority of strokes in both races are due to cerebral infarction rather than to cerebral hemorrhage.

## Hypertensive End-Stage Renal Disease


#### Abstract

N. \# End-stage renal disease caused by hypertension occurs much more $\#$ * commonly in Blacks than in whites. Blacks with hypertension are * at much greater risk of developing end-stage renal disease than $*$ * whites. Blacks with hypertensive end-stage renal disease treated * * with dialysis have a more favorable cardiovascular mortality outlook than whites.




## (i) Introduction

End-stage renal disease (ESRD) is one of the sequelae of hypertension. Data collected by the National Center for Health Statistics(41) indicate that hypertensive heart and kidney disease, and hypertensive kidney disease together account for $23.6 \%$ of all deaths related to kidney and urinary tract disorders.

## (ii) Morbidity

Primary hypertensive disease accounts for $13 \%$ of patients on maintenance dialysis. In 1976 the total population undergoing end-stage renal disease treatment was about $31,000,70.5 \%$ white and $24.2 \%$ Black. (41) In 1982, the total population of Medicare ESRD dialysis patients was $56,046: 66 \%$ were white, and $30 \%$ were Black. (42) Hypertension was the cause of ESRD in $27.7 \%$ of the Black patients and $11.9 \%$ of the white, so that the total number of such Black patients actually exceeded that of whites [Table 16]. The proportion of Blacks is about double that expected from the $11-12 \%$ of the population which is Black, and attests to either or both a higher incidence and a greater duration of kidney failure in the Black population.

## (iii) Incidence

Studies by Easterling(43), 1977, in southeastern Michigan confirmed that the incidence of end-stage renal disease is three times higher in the Black population than in the white, due to the higher prevalence of glomerulonephritis, and hypertensive and diabetic nephropathy in the Black population. In particular, Blacks were 16.9 times as likely to develop renal disease from hypertension. An almost identical result for the risk of hypertensive renal disease was obtained in a study in Jefferson County, Alabama in the 1974-78 period. (44) In this study, the risk of end-stage renal disease was four times higher in Blacks than in whites. The yearly incidence of hypertension-related end-stage renal disease was 6.4/100,000 in Blacks compared to $0.36 / 100,000$ in whites (relative risk: 17.8).

The most comprehensive, published data are those provided by Sugimoto and Rosansky (45) for the incidence of treated end-stage renal disease in 20 contiguous eastern states in the period 1973-79.

For white men and women, the incidence rates are 60 and $40 / \mathrm{million}$ person-years, respectively, since 1977, and the incidence rates in Blacks in 1979 were $125 /$ million person-years in men and $100 / \mathrm{million}$ person-years in women. The incidence of treated end-stage renal disease was twice as high in Blacks as in whites, and hypertensive nephropathy was seven times as common in Blacks as in whites [Figure 6]. The hypertensive nephropathy rates for Blacks were $35 / \mathrm{million}$ person-years for men and $24 /$ million person-years for women. These constitute $28 \%$ and $24 \%$, respectively, of the total end-stage renal disease rates in 1979. Although it has been shown that hypertension control reduces end-organ damage, it is noteworthy that end-stage renal disease incidence rates were still increasing in both Black men and women during the late 1970s, a period in which hypertension control activities are thought to have been more effective.(26) The explanation for this is unclear.

## (iv) Mortality

Survival of Blacks and whites undergoing dialysis or transplantation has been compared. In general, among patients receiving dialysis, Blacks had lower death rates than whites, but the rates were comparable among patients who underwent transplantation. It should be noted as well that Blacks undergoing dialysis are likely to be younger, $41 \%$ being under 50 years of age compared to $35.5 \%$ for whites.(46) For 1982, data on Medicare ESRD dialysis stations showed that mortality rates for hypertension-related ESRD in Blacks were $57 \%$ of those in whites. (42) The prognosis of patients undergoing dialysis for hypertensive renal disease is significantly worse than for those undergoing dialysis for end-stage renal disease associated with polycystic kidneys or glomerulonephritis, but better than those undergoing dialysis because of diabetic nephropathy.

## II: EXPLANATIONS FOR DIFFERENCES



* Major socioeconomic differentials exist between Blacks and *
* whites and affect their respective life experiences, biological *
\% risk factor distributions, and access to medical care. A simple *
* biomedical model is thus probably inadequate for assessment of *
* Black-white differences in cardiovascular disease morbidity *
* and mortality. A more comprehensive analytic strategy that *
* takes account of the social context of disease is required to *
$\star$ clarify the causes of Black/white CVD differences. $\quad$.
以


## Introduction

It must be stated clearly at the outset that the currently available data are inadequate to allow definitive explanations of the differences in the cardiovascular disease (CVD) experience and outcomes of the Black and white populations.(2) Hypertension and
hypertension-related diseases - in particular, stroke - have been studied more vigorously, and the treatment of hypertension has been the focus of major efforts in the last decade.

In attempts to explain Black-white differences in cardiovascular disease morbidity and mortality, it is appropriate to examine and compare, in Black and white populations, the impact of:

1. Biologic, and/or physiologic variables affecting coronary heart disease, hypertension, and hypertension-related diseases.
2. Socioeconomic factors as they influence risk, incidence, prevalence, and severity of disease.
3. Behavioral and/or cultural variables such as health beliefs, health practices, and health-seeking and health-relevant behaviors.
4. Issues related to access to and utilization of the health care system.
In view of the persistent social disadvantage of U.S. Blacks, a circumscribed biomedical view which focuses on biological and physiological factors independent of the context in which they occur would result in selective inattention to the social causation of disease. (47) Though satisfactory links between these different levels of analysis have not been established, a synthesis of the conclusions from these different levels of analysis is clearly required. Such a synthesis is essential for the formulation of interventions intended to eliminate these Black-white CVD differentials.

In this analysis, as in the earlier sections, coronary heart disease, hypertension, stroke, and hypertension-related renal disease will be dealt with in that order.

## A: Biologic and/or Physiologic Variables

\%

* Because of the paucity of studies of coronary disease in Blacks, \#
$\star$ the data on the impact in Blacks of biological risk factors for
* CHD, identified in white populations, are inadequate. These de* ficiencies may cause coronary disease prevention programs de* signed for Blacks to be less effective than expected.


## Coronary Heart Disease

(i) Introduction

The data on risk factors for coronary heart disease in Black populations were reviewed in 1982 by Gillum and Grant. (48) These authors noted that, though certain physiologic characteristics such as hypertension, hypercholesterolemia, low levels of high-density lipoprotein cholesterol, cigarette smoking, diabetes mellitus, and gender are considered independent predictors of coronary heart disease in white populations, there is a dearth of studies of CHD incidence in Black populations that examine the impact of the putative CHD risk factors. Since that review, more data have become available on the association of some of these risk factors with coronary heart disease incidence and mortality in Black populations
in the United States. However, the data remain inadequate, and this may adversely affect the design of appropriate prevention programs and educational interventions for the Black community. In this discussion of the CHD risks associated with these biological risk factors and their prevalence, data from population-based studies, especially recent ones, are given preference.
(ii) Age, Sex, and Family History

As in whites, CHD mortality, incidence, and prevalence increase with age. (48) The lower prevalence of ECG evidence of healed myocardial infarction in Blacks over the age of 65 years(10) may reflect increased mortality at younger ages from all causes, as well as a higher Black case-fatality from acute myocardial infarction. Black men are at higher risk of CHD than Black women, but in view of the small sex mortality difference, Black women appear to be at higher risk than would be predicted from their gender on the basis of data on whites. Aggregation of CHD within families has not been reported for Black populations, though some small studies have reported aggregation of risk factors in Black families.(49)
(iii) Hypertension
\%

* The impact of hypertension on the development of coronary disease* * in Blacks may be somewhat less than in whites. However, hyper- * * tensive Blacks who develop coronary disease appear to be at par- ${ }^{*}$ * ticularly high risk of death. Vigorous treatment of hypertension * * has a similar impact on reducing CHD incidence in Blacks and * * whites.
$\stackrel{\rightharpoonup}{*}$


Current data on population blood pressure levels and the prevalence of elevated blood pressures and hypertension in Blacks have been presented earlier.

Population-based data on the impact of hypertension on CHD incidence and mortality in Blacks are available from the Evans County, Georgia study which began in 1960 ( $71 / 4$-year follow-up for incidence(13), 20-year follow-up for mortality(14)) and from the 5 -year follow-up, beginning in 1973, of men screened for participation in the Multiple Risk Factor Intervention Trial.(34) Because of the small number of cases in the Evans County incidence study, the statistical power was limited. In Evans County Black men(13), but not in Black women, age-adjusted incidence of CHD increased with blood pressure level. In addition, at each blood pressure level, CHD rates were lower in Black than in white men, but similar in Black and white women. In the Evans County 20-year mortality study(14) of Black men ages 40 to 64 years, there were 31 deaths attributed to CHD among 294 examinees. Blood pressure had a "monotonic, strong, highly statistically significant association with CHD mortality in middle-aged Black males" [Table 17].(14) In the MRFIT screenee follow-up(34), $23.7 \%$ of the 450 deaths in Black men
were attributed to CHD (ICD 410-414), compared to $37.1 \%$ of the 4,602 deaths in white men (a significant difference); $72 \%$ of the CHD deaths in each racial group were coded as ICD 410, or acute myocardial infarction [Table 18]. The age-adjusted logistic regression coefficients for the asssociation between diastolic blood pressure and CHD deaths did not differ significantly between Black and white men [Table 19], but it was noted that the difference in CHD rates in Black and white men occurred primarily in the upper quintile of diastolic blood pressure ( $>91 \mathrm{~mm} \mathrm{Hg}$ ) [Figure 7]. In this group, the age-adjusted rate was $8.4 / 1000$ for white men and $5.2 / 1000$ for Black men. Black hypertensive men appeared to be at lower risk of CHD mortality than white hypertensive men in this study.

The data on the impact of hypertension on CHD are conflicting. Data from two clinical trials suggest higher CHD mortality rates for Black subjects who have both hypertension and CHD. In the Hypertension and Detection Follow-up Program (HDFP) (12), the 5-year mortality rates among Black men with angina pectoris and with ECG-MI at baseline were 1.8 and 2.0 times as high, respectively, as the rates among similar white men. In addition, in the Beta-Blocker Heart Attack Trial(50), the mortality rate in placebo-treated Black men was significantly higher than in placebo-treated white men. One feature of the Black group was a significantly higher prevalence of a history of hypertension (57\%), compared to that for non-whites (39\%). This characteristic, as well as a significantly higher prevalence of smoking might account for the observed mortality difference.

Race-sex-specific incidence rates for stepped care participants in the HDFP(12) show only small differences in 5-year incidence of CHD (ECG MI, or history of MI, or MI by Rose Questionnaire) between Blacks and whites: rates of MI, diagnosed by ECG alone, were higher in Black men and women than in white men and women respectively. No specific attempt has been made to ascertain whether the impact of blood pressure on CHD incidence differed between the racial groups in this vigorously treated population. However, there is evidence that vigorous stepped care treatment of hypertension nearly equalized the risk of CHD between the races(51), since, in comparison with the referred care group, similar reductions in 5-year incidence of fatal CHD and nonfatal MI were observed in Blacks and whites.

* Mean serum cholesterol levels in Black and white adults are sim- * * ilar and some data suggest similar effects of cholesterol on cor- ${ }^{*}$


## (a) Total Blood Cholesterol

Total blood cholesterol distributions and the prevalence of values in excess of arbitrarily chosen limits have been studied in Black populations in the United States. The Health Examination Survey of 1960-62(52) reported that the age-adjusted mean serum cholesterol levels of Black men and women were 210.8 and $214.0 \mathrm{mg} / \mathrm{dl}$, both lower than those reported for white men and women, 217.4 and $224.1 \mathrm{mg} / \mathrm{dl}$, respectively. The Health and Nutrition Examination Survey of 1971-74 (NHANES I)(53) detected no consistent differences between Black and white adult men in cholesterol means or distributions. The same is true for women, except that Black women, ages 55-64 years, had slightly higher levels than white women.

In NHANES I(54), serum cholesterol level was related to body mass index, but the association of higher serum cholesterol levels with higher body mass index was less consistent in Black men than in the other groups. In addition, within each quintile of body mass index, the mean serum cholesterol level was lower in Black women than in white [Figure 8].

Data on the prevalence of elevated serum cholesterol levels, defined as levels of at least $260 \mathrm{mg} / \mathrm{dl}$, have been published for Blacks examined in NHANES I and NHANES II. Between survey periods, there was a decrease in the age-adjusted prevalence of elevated serum cholesterol levels in Black adults, but the decrease was not statistically significant.(55)

## (b) High-Density Lipoprotein Cholesterol

An inverse relation between plasma high-density lipoproteins and CHD risk has been detected in several white populations. In some population-based studies, Blacks have been observed to have higher high-density lipoprotein (HDL) levels and lower levels of low-density lipoprotein (LDL) cholesterol.(56) This has been found in pediatric and adolescent, as well as adult populations [Table 20]. In the Lipid Research Clinics Study which included only a small number of Blacks ( 424 patients), the HDL levels were significantly higher and LDL levels significantly lower in Blacks than in whites. (57) The
differences are larger in men than in women, and in some studies of adult women, no Black excess was detected. (56) HDL levels are affected by a number of environmental factors. Significant direct relationships with physical activity and alcohol intake, and significant inverse relationships with measures of body mass index, cigarette smoking, and use of some antihypertensive agents have been detected. Some authors have speculated that the absence of significant differences between adult Black women and white women might be due to the high prevalence of obesity in Black women. In addition, because statistical adjustment for the environmental variables examined do not eliminate Black-white differences, there has been speculation that there is an inherent, possibly genetic, tendency for Blacks to have higher HDL-cholesterol levels. (58) It has been suggested that elevated HDL levels might account for the lower incidence of CHD in Blacks in some studies (59), but no analysis addressing this issue has yet been published.

As in the case of blood pressure, few studies have assessed the relationship between serum cholesterol and CHD incidence in Blacks. In the Evans County incidence study (60), the interactive effect of cholesterol and age was significantly related to CHD incidence in white men, but the logistic function predicted an incidence of CHD in Blacks far in excess of that observed. The investigators concluded that Blacks do respond to the standard risk factors (including cholesterol) similarly to whites, but with a lower level of CHD than would be expected, given the levels of the risk factors. In the $20-y e a r ~ E v a n s ~ C o u n t y ~ m o r t a l i t y ~ s t u d y(14), ~ c h o l e s t e r o l ~ w a s ~$ significantly related to CHD mortality in middle-aged Black men [Table 17]. In the MRFIT screenee five-year mortality follow-up study (34), the age-adjusted CHD mortality rates for Black and white men were similar in each quintile of serum cholesterol concentration [Figure 9], and the logistic regression coefficients were similar for white and Black participants, 0.0071 for Black men and 0.0079 for white men.
(v) Cigarette Smoking


* The prevalence of cigarette smoking is greater in Black than in * * white men, but the prevalence of heavy smoking is greater in * * white than in Black adults. In recent years, there has been a re-* * duction in the proportion of Black adults who are light or mod- * * erate smokers. Smoking appears to increase the risk of coronary * * disease mortality similarly in Blacks and in whites. *
* 

National probability estimates of the prevalence of cigarette smoking in Black adult men and women in the period 1965 to 1983 are available from the National Health Interview Survey (61) and the National Health Examination Surveys, NHANES I and II.(55) Age-adjusted national estimates of the percentage of current smokers indicate an $8-9 \%$ higher prevalence of smoking in Black compared to white men over the age of 20 years in 1965,1976 , and 1980. $(62,63)$

Age-specific tabulations indicate that the prevalence of smoking is high in 25-44 year-old men [Table 21]. In addition, the Black excess in prevalence is observed in every age-group in the NHIS and NHANES I and II data [Table 22]. On the other hand, the prevalence of heavy smoking (25 or more cigarettes per day) is substantially higher among white than Black men, and the rate of increase in heavy smoking rates between 1965 and 1980 was greater for white men. The age-specific data indicate that 35-64 year-old smokers are more likely to be heavy smokers than the other age-groups, but this is common to both Black and white men.

Smoking patterns for women are very different from those of men. Rates are lower and consistent Black-white differentials are not observed. The age-specific data for women indicate a substantially lower prevalence of smoking in women 65 years and older compared to younger women. Heavy smoking is also more common among white women than Black women and, as among men, the rate of increase in the prevalence of heavy smoking between 1965 and 1980 was greater for white than for Black women.

Between NHANES I and NHANES II, the proportion of Black people who currently smoke cigarettes decreased [Table 21]. The decline was greater for women than for men, 15 percentage points compared to five. The proportion of Black adults who smoked 25 or more cigarettes per day did not change significantly. Thus, the decrease in the proportion of smokers between NHANES I and NHANES II was due to a reduction in the proportion of Black adults who were light or moderate smokers.

The impact of cigarette smoking on CHD risk in Black subjects has been examined in a small number of studies. A history of cigarette smoking was a significant predictor of $71 / 4$-year CHD incidence in whites in the Evans County study and appeared to affect Blacks as well.(60) A history of current smoking was a significant independent predictor of death attributed to CHD in Black men in the 20-year Evans County follow-up.(14) In the MRFIT screenee follow-up, (34) the 5 -year age-adjusted CHD mortality rates were very similar for Blacks and whites at different levels of cigarette consumption, except for those who smoked 26-35 cigarettes per day [Figure 10]. The logistic regression coefficients for Black and white men did not differ significantly from each other, and reflect a significant positive association between cigarette smoking and CHD mortality [Table 23]. In the American Cancer Society prospective study (64) of one million Americans followed for 12 years (1960-72), about 25,000 Blacks were enrolled. CHD mortality ratios in subjects grouped according to the number of cigarettes smoked were similar at given smoking levels in Black and white men, and slightly lower in Black than in white women. There was evidence of an enhanced effect of smoking on the risk of CHD death in individuals with a history of high blood pressure or other cardiovascular disease [Figure 11].

年
$\therefore$ The prevalence of diabetes mellitus, both diagnosed and un- *

* diagnosed, is greater in Blacks than in whites. The effect of $\quad$ *
* diabetes mellitus on coronary disease in Blacks has not been *
* assessed adequately. $\quad \pm$
* 



Though all-cause mortality rates in non-white diabetics in the U.S. are twice those of whites, the impact of diabetes on CHD has been examined in few studies.(48) The prevalence of diabetes is higher in Blacks than in whites. In the Health Interview Survey, 1964-1965(65), the self-reported prevalence of diabetes in non-whites, 13.3/1000, was similar to that in whites, 12.1/1000. In the Health Interview Survey of 1973(66), the self-reported prevalence was 23.9/1000 for non-whites and 19.9/1000 for whites. More recently, NHANES II data on subjects, ages 20-74 years, reveal a higher prevalence in Blacks than in whites of a medical history of diabetes (self-report of a physician diagnosis and current or past use of diabetic therapy), $5.2 \%$ compared to $3.2 \%$; and of undiagnosed diabetes, $4.4 \%$ compared to $3.0 \%$ [Table 24]. These differences are not statistically significant. Cooper et al(67) have reported that, in an employed Chicago population the prevalence of diabetes was approximately $3 \%$ in men of both races and $2 \%$ in women. Moreover, plasma glucose levels one hour after a 50 gram glucose load were lower in women than in men, and in Blacks than in whites. Black women had the lowest levels despite having the highest prevalence of obesity (relative weight>1.45), $21.1 \%$. These data are consistent with those reported by a Kaiser-Permanente program(68) which studied 12,000 Blacks and 88,000 whites between 1964 and 1968. In this study, mean serum glucose levels one hour after a 50 -gram glucose load remained significantly lower in Blacks than in whites after adjustment for height, weight, ponderal index, and triceps skinfold thickness. Such data suggest that Black women, even obese Black women, may be less prone to diabetes. However, the markedly high prevalence of obesity in Black women probably contributes to the excess prevalence of a medical history of diabetes as, for example, in 55- to 64 -year-old Black women in NHANES II: $16.3 \%$ compared to $6.6 \%$ in white women of similar age.

The Chicago investigators(67) observed that cardiovascular death rates in Black men with diabetes or hyperglycemia were slightly lower than those in white men. However, compared to normoglycemic people, Black men with diabetes or hyperglycemia had a relative risk of cardiovascular death of 1.43 . It should be noted that these rate comparisons are based on small numbers of deaths.

In the MRFIT screenees $35-57$ years of age(69), $1.5 \%$ of those men without a history of heart attack were being treated with medications for diabetes; $0.2 \%$ were Black and $1.3 \%$ non-Black. Age-adjusted . 5 -year CHD death rates among Black diabetics were more than twice those among Black nondiabetics, 8.5/1000 compared to 4.1/1000.

The comparable rates among non-Black diabetics were $14.2 / 1000$ and 4.3/1000 which yields a relative risk of greater than 3 . However, these analyses are based on only 7 CHD deaths among Black diabetics, a $1 \%$ mortality rate [Table 25].

## (vii) Electrocardiographic Abnormality

## 

* Electrocardiographic abnormalities are predictive of CHD in *
* some studies of white patients. Such abnormalities are more $\quad \div$
* common in Blacks than in whites. The impact of these abnormal- $\quad *$
* ities, especially electrocardiographic evidence of left ventric- *
$\star$ ular hypertrophy (ECG-LVH), has not been assessed satisfactorily $*$
$\pm$ in Black populations. $\quad$.
K
In studies of white populations, electrocardiographic abnormalities -
in particular ST depression, major T-wave abnormalities, and
increased QRS voltage - have been associated with an increased incidence of CHD. (70) In the Framingham study (71), electrocardiographic evidence of left ventricular hypertrophy (ECG-LVH) was associated with a three-fold increased risk of CHD after adjustment for the effect of elevated blood pressure. A number of population-based studies, including the Evans County study(60), and the Birmingham Stroke Study (72) have shown such ECG abnormalities to be more common in Blacks than in whites. In Evans County(73), the age-specific prevalence of ECG-LVH (Minnesota Code 3.1) was 2-3 times higher in Blacks than in whites, and even when the rates were adjusted for differences in age, blood pressure, body habitus, habitual physical activities, and smoking habits, the Black/white differences persisted. Similarly, in the Birmingham Stroke Study (72), the contribution of race to increased R-wave amplitude was shown by multiple regression analysis to be independent of blood pressure, a history of treated hypertension, or a history of angina pectoris or prior myocardial infarction.

In the Evans County study(74), white men had a markedly higher incidence than Black men of new coronary events in the presence of ECG abnormalities. Each of the abnormalities carried an increased risk of coronary disease for white men, but not for Black men. Black women had increased CHD incidence rates only with left axis deviation.

No published studies to date have revealed a statistically significant excess risk of CHD incidence in Blacks associated with ECG-LVH. In the Evans County Study (73), ECG-LVH in the presence of hypertension was associated with an excess (not statistically significant) risk of death over 9-12-year follow-up in all four race-sex groups. However, in the HDFP referred care men(75), ages 40-69 years, with diastolic blood pressures of $90-104 \mathrm{~mm} \mathrm{Hg}$, and similar educational attainment (less than high school), the age-adjusted all-cause mortality rates were significantly higher in both Blacks and whites with ECG-LVH than in those without. The relative risk was lower in Blacks (2.1) than in whites (2.7) [Table

26]. The prognostic implications of LVH for CHD in Blacks are still in need of clarification. Analysis of data sets that include ECGs and echocardiograms should be of value in assessing the prognostic implications of anatomic LVH.

## (viii) Obesity


$\therefore$ Obesity is especially common in Black women and may provide a $\dot{*}$
$\therefore \quad$ partial explanation for their excess coronary disease risk. *


Obesity is prevalent in Black women. The Health Examination Survey of 1960-62(76) found that despite similar heights in Blacks and whites, the proportion of the population weighing 170 pounds or greater was 38.9\% for Black men, 30.3\% for Black women, $45.4 \%$ for white men and $14.3 \%$ for white women. In NHANES I(77), there was little difference in height or weight between Black and white men, but the mean weights and 90 th percentiles were significantly higher for Black women than for white women. The entire weight distribution for Black women was shifted towards higher weights [Figure 12]. Comparison of the NHES and NHANES I data indicates that Black women less than 44 years of age weighed more at the latter examination. NHANES II data(58) revealed that the Quetelet index (weight, divided by [height squared]) was higher in Black than in white women, but similar or somewhat lower in Black than in white men. In the three age-groups 21-45 years, 45-65 years and over 65 years, Quetelet indices were considerably higher in Black than in white women, but not significantly different for Black and white men. In addition, maximum self-reported weight, minimum weight, and weight at age 25 years were higher in Black than white women, but comparable in Black and white men [Table 27].

It has been speculated that pandemic obesity might explain the apparently higher CHD mortality in Black than in white women, perhaps by means of an association with lower HDL-cholesterol levels. (58) The evidence presented earlier suggests that the higher prevalence of diabetes mellitus in middle-aged Black women may be related to obesity, and an association between elevated blood pressure and obesity has also been reported. The Evans County investigators reported a 2.2 -fold higher incidence of CHD in Black men in the upper tertile of Quetelet index compared to those in the lower tertile. (78) The age-adjusted rate in white men in the upper tertile was still four times greater than that of Black men in that tertile. No report was provided of the impact of obesity on CHD risk in women. No significant independent relationship of Quetelet index to 20-year CHD mortality was observed in Evans County Black men. (14)
$\star$ tension control should decrease coronary disease incidence and $\Rightarrow$

In view of the excess of hypertension in Blacks (granted the similarity in serum cholesterol levels, and the overall similarity in cigarette consumption in Blacks and whites), it can well be asked why are not CHD rates for Blacks higher than they are? In fact, CHD mortality rates for Black women exceed those for white women. It is unknown whether obesity and effects mediated via lower: HDL-cholesterol levels and elevated blood pressure levels might account for this, although this is a plausible explanation. It has been speculated that there might be measured or unmeasured risk factors other than hypercholesterolemia, hypertension, and cigarette smoking which might protect Black men against further elevation of CHD rates. (60) HDL-cholesterol may be the major protective factor(59), but this hypothesis has not been examined in large prospective studies that included adequate numbers of Black and white men. Physical activity(60) associated with occupation has been suggested as a factor which might protect Black men against CHD. This effect might be mediated by means of lower blood pressure, lower body weight, and higher HDL-cholesterol levels. However, the data to test this hypothesis do not exist. Some studies have suggested that differences in a number of hemostatic variables between Blacks and whites, such as higher fibrinolytic activity, and longer platelet survival in Blacks might protect them against the development of coronary atherosclerotic lesions and coronary thrombosis.(8) Some of these factors are thought to be affected by diet and physical activity levels. Long-term studies in this area have recently been sponsored by the NHLBI.

It has already been noted that the multiple logistic risk function derived by the Evans County investigators suggested that elevated serum cholesterol, hypertension, and cigarette smoking all increased CHD risk in Blacks, but to a lesser degree than in whites. (60) In view of the low incidence of CHD in the Evans County population, the generality of these findings to the U.S. population cannot be assumed. However, the consistency between racial groups of the logistic regression coefficients for the association of blood pressure, cigarette smoking, and serum cholesterol with CHD deaths in
the MRFIT screenee five-year follow-up study is striking. These data derived from studies of 23,490 Black men and 325,384 white men in 18 U.S. cities from 1973 are perhaps the most reliable data available on the impact of these risk factors on CHD mortality in Blacks.
However, it must be re-emphasized that these analyses and the 20 -year Evans County analyses employ possibly unreliable death certificate diagnoses of CHD. These data raise the provocative suggestion that the impact of blood pressure on CHD mortality is less in Black hypertensives than in whites. The MRFIT investigators noted that the Black/white relative risk of CHD death (after adjustment for age, serum cholesterol concentration, and cigarettes smoked per day by logistic regression) was 1.15 for men with diastolic blood pressures less than 91 mm Hg , and 0.69 for those men with higher diastolic blood pressures. This difference was statistically significant. However, as noted earlier, a history of hypertension in Black men with CHD is hardly a benign finding.

Rowland and Fulwood(55) have presented an analysis of the changes in risk factors that took place in Blacks and whites from NHANES I to NHANES II, (the period from 1971-75 to 1976-80), and have attempted to correlate these changes with the observed CHD mortality rates for adults, Black and white, 35-74 years of age, during this period. In the absence of a risk equation derived from observation of a large Black cohort, they used a multiple logistic function, based on observations in the predominantly white Framingham Study, and estimated that the greater decline in elevated blood pressure and cigarette smoking in Blacks during this period could account for the greater estimated decline in expected CHD mortality for Blacks compared to whites: $13 \%$ and $16 \%$ for Black men and women; $7 \%$ and $8 \%$ for white men and women [Table 28]. The observed mortality declines corresponded more closely to those expected for Blacks than they did for whites. In the absence of more reliable data, public policy and clinical practice for primary prevention of CHD in Blacks should be based on these observations and analyses.

Hypertension
(i) Introduction

In 1979, Gillum(79) reviewed the literature on racial blood pressure differences in the United States. He concluded that the cause of these differences remains speculative, and noted that racial differences in renal physiology and environmental influences such as socioeconomic status are likely candidates for important contributions to blood pressure differences. In the CHD risk factor review published in 1982, Gillum and Grant (48) asserted that selected studies published since this initial review have added nothing to contradict its conclusions.

## (象

* "Black" in the United States is a sociological category. Some *
* investigators have confused ethnic identity with genetic con- *
* stitution, simplistically equating them. The heterogeneity of *
$\pm$ blood pressure levels and hypertension prevalence in Black pop-
* ulations in Africa, the Caribbean, and the Americas casts doubt * on the proposition that genetic factors are primarily respon* sible for the blood pressure excess in U.S. Blacks.


Many hypotheses have been advanced to explain the higher prevalence of hypertension in U.S. Blacks. Among them are theories that have in common the postulate that the tendency of Blacks to develop excessively elevated blood pressure has a genetic basis. If so, the genetic predisposition to elevated blood pressure should be a characteristic common to Black-skinned people everywhere. Thus, Black people in Africa and elsewhere should share approximately the same rate and severity of hypertension. However, when the available epidemiologic data on blood pressure levels in Black populations in Africa, the Caribbean, and the Americas are examined, it becomes clear that any explanation of blood pressure differences between Black populations must take explicit account of environmental determinants and influences.

Though Blacks in the Caribbean and the Americas are mostly of West African descent, an extensive process of miscegenation occurred during and subsequent to the period of slavery(80), so that Black populations in these regions are racially heterogeneous. In the United States, the term "Black" encompasses an identifiable, visible, ethnic group, with a distinctive historical, social, and economic experience. This creates significant problems for the interpretation of racial data on blood pressure differences since these differences have important socioeconomic and psychosocial correlates. Moreover, since "Black" is a sociological category, it does not describe a group with unequivocally identifiable or uniform genetic characteristics. In scientific investigations of blood pressure distributions in the U.S. Black population, the racial heterogeneity that is characteristic of this ethnic group is, typically, not even assessed. (8) Moreover, assessment of the degree of admixture between genes of presumed $(81,82)$ Black and white racial origin relies on a number of unverifiable assumptions.(83)
(iii) Blood Pressure Distributions in African and Other Black Populations

Examination of published data on blood pressure in African populations since the 1920 s reveals marked diversity. In particular, hypertension has been shown to be rare in many rural communities. in Kenya(84), Uganda(85), Tanzania(86), and the Gambia.(87) Large-scale epidemiologic surveys in Ghana, West Africa(88) have reported little
rise in mean systolic blood pressure with age among rural subjects of both sexes, and a rate of increase that was more gradual among rural subjects than among urban subjects or than among a sample of 827 Black Americans examined in the NHES in 1960-62.(28) It is particularly noteworthy that systolic blood pressures of rural Ghanaians of most ages were $20-25 \mathrm{~mm} \mathrm{Hg}$ lower than those of urban Ghanaians and U.S. Blacks, and that the difference for diastolic blood pressure was $10-15 \mathrm{~mm} \mathrm{Hg}$. Only a small percentage of rural Ghanaians had diastolic blood pressures in excess of 95 mm Hg , and this occurred only in subjects older than 40 years of age, some 15 years earlier than the transition occurs in the United States. It is also significant that the age-adjusted prevalence of hypertension among Zulu men in Durban, South Africa was $23 \%$, almost identical to the $22 \%$ prevalence among white men. (89)

In the Caribbean(90-94), despite similarities of racial and cultural background among the Black populations in different islands, there is marked heterogeneity of blood pressure levels and hypertension prevalence. For example, the mean systolic blood pressure among men in a 1981 population sample in the island of St. Lucia(94) was 20 mm Hg lower than that observed in a similar population sample in the Island of St. Kitts(91) in 1958.

It is in the context of such information on blood pressure distributions in Black populations outside the U.S. that genetic explanations of blood pressure differences between Blacks and whites in the U.S. should be considered. Specifically, explanations of blood pressure differences among Black populations must take explicit account of environmental determinants.

## (iv) Differential Sensitivity to Dietary Electrolytes?

为
$\pm$ Some data suggest that there are differences in renal sodium $\quad \pm$
$\stackrel{\star}{*}$ handling between Blacks and whites, but the data do not indicate $\#$
$\therefore$ that higher sodium intake in Blacks explains their higher pre-
$\star$ valence of hypertension. National data show that potassium in-

* intake is lower in Blacks. This may confer greater sensitivity
* to the effect of sodium and result in higher blood pressures.
$\pm$ Currently available data on cell membrane electrolyte transpart $\quad *$
* systems do not explain Black/white blood pressure differences. *

人
There is some evidence that differences in renal physiology may account for some blood pressure differences between "Blacks" and whites. It has been suggested that hypertension is the consequence of an inherited renal defect in sodium excretion which confers an increased sensitivity of blood pressure to sodium intake. Luft and coworkers(95) provided laboratory evidence of differential sensitivity to the effect of sodium intake in humans, and of differences in sodium handling between Blacks and whites with normal blood pressures. Their analysis of duplicate dietary collections from Blacks and whites in Evans County, Georgia(96) did not provide evidence that the higher prevalence of hypertension in Blacks in this
area could be attributed to a greater dietary intake of sodium. These investigators noted that, although Blacks and whites ingested similar quantities of sodium, dietary potassium intake for Blacks was consistently less than that for whites. Similar conclusions have been reached from dietary recall studies in other U.S. populations (97) and in NHANES I. (98) Though dietary recall methods are less accurate than analysis of duplicate dietary collection, it has been speculated that the excess of high blood pressure in Blacks might be related to relative potassium deficiency and the impact of this on renal sodium handling. (99). One fairly consistent observation in the area of renal physiology has been that suppressed plasma renin activity is found more commonly in U.S. Blacks. $(79,100)$ However, this is not a universal observation, since studies in a group of "Black" Jamaican hypertensives revealed a prevalence of low-, normal-, and high-renin groups that was $31 \%, 45 \%$, and $24 \%$ respectively.(101)

More recently, evidence to support a genetic hypothesis has been sought by examination of the activity of cell membrane transport systems for electrolytes and intracellular electrolyte concentrations in Black and white subjects. Blaustein(102) has recently reviewed the available data and concluded that they give little cause for hope that the key to Black-white differences will be found therein. Trevisan et al(103) have concluded that the pattern of sodium countertransport across erythrocyte membranes in race-sex groups is "not consistent with a direct relationship between countertransport or sodium concentration and blood pressure which applies across both racial groups". Ringell et al(104) have concluded that furosemide-sensitive sodium and potassium cotransports and intracellular sodium content are not clinically useful in the identification of essential hypertension in Black men because of substantial overlap in observed values between hypertensive and nonhypertensive men. Tuck et al(105) also could not find sodium-potassium cotransport assay useful in identifying hypertension-prone Black normotensive subjects.
(v) Correlates of Hypertension Incidence: Implications for Primary Prevention

Observations of Black populations in both rural and urban areas show correlations between the incidence of hypertension and some physiological factors, which suggest possible approaches to primary prevention. In Black women, ages 15-29 years, in Evans County(106), the seven-year incidence of elevated blood pressure was associated with weight gain. A similar observation was made in Black women, ages 30-69 years, in an inner-city (Baltimore) population. (107) Reported change in weight was a significant predictor of elevated pressure. These findings indicate the importance of weight control for hypertension prevention in Black women. In addition, Voors et al(108) have shown that potassium administration produced marked natriuresis and decreased blood pressure levels in Blacks. Such data suggest that, at levels of potassium intake similar to those observed in Whites, blood pressure levels in Blacks would be lower.

## (i) Hypertension

The major risk factor for stroke is hypertension. Since the prevalence of hypertension is significantly higher in Blacks than in whites, this accounts in part for the Black excess of stroke incidence and mortality. In the MRFIT screenee follow-up study (34), the logistic regression coefficient for the relationship between diastolic blood pressure and death from cerebrovascular disease in Black men was significantly higher than for white men [Table 19, Figure 13]. In the Framingham study(109), the dominant predictors of stroke risk were blood pressure level and ECG-LVH. In the Hypertension Detection and Follow-up Program(38), mortality rates, including those for stroke, were increased among men with ECG-LVH by Minnesota Code criteria. In the Evans County study, the presence of any ECG abnormality was associated with a slightly higher risk of stroke in both Black men and women. (110) Thus, Blacks with hypertension and evidence of ECG abnormalities are at higher risk of stroke.

## (ii) Cholesterol and Cigarette Smoking

In the Evans County Study(110), there was no consistent relationship between the serum cholesterol level and risk of stroke among Blacks or whites. No consistent relationship was noted between age-adjusted cerebrovascular disease mortality rates by cholesterol quintiles in the MRFIT screenee follow-up study [Figure 14].(34) Similarly, the logistic regression coefficient relating number of cigarettes smoked per day to death from cerebrovascular disease for Black men indicated no significant association.
(iii) Conclusion

The major factor which accounts for the Black-white disparity in stroke incidence, morbidity, and mortality is probably hypertension. Since stroke mortality rates have been declining since the 1930s, but have declined even more rapidly in the late 1970s, it is likely that part of the increased rate of decline in stroke mortality in Blacks is the result of improved hypertension control. (111)

## Hypertensive End-Stage Renal Disease

The higher prevalence of hypertension in Blacks can be invoked to explain much of the disparity in incidence rates of end-stage renal disease (ESRD). Blacks have a disproportionately high rate of renal failure from hypertensive disease, and though the diagnoses in the studies reported ( 43,440 patients) were based on clinical rather than histological evidence, diagnostic error could not totally explain the 17-18 fold disparity. No explanation, other than a greater
prevalence and severity of hypertension especially in Black women in the fourth decade of life has been proposed. It appears likely that earlier recognition and more vigorous treatment of hypertension might reduce the incidence of ESRD in Blacks: the continued increase in Black ESRD incidence in the eastern U.S.A. until 1979 may represent a cohort effect.

White subjects with ESRD have a higher risk of cardiovascular mortality and, possibly, of CHD than Blacks, probably due to enhancement of the rate of progression of atherosclerosis. (112) Racial differences in HDL-cholesterol levels between Blacks and whites with ESRD may account for lower CHD incidence and mortality rates in Black subjects with ESRD.(113)

Conclusion
(i) The Role of Hypertension in Black CVD Mortality and Morbidity

The analyses presented suggest a central role for hypertension, both as a major cardiovascular disorder and as an explanation of enhanced individual risk, if not of CHD, at least of stroke and ESRD in Blacks. Even if the risk of CHD in hypertensive Black males is somewhat less than in hypertensive white males, as the MRFIT screenee data suggest, the impact of hypertension on CHD risk in the Black population as a whole, remains sizeable because of the high prevalence of hypertension.
(ii) Racial Trends in Hypertension-Related Mortality: Role of Hypertension Treatment
(
$\pm$ Stroke mortality has declined more rapidly in recent years than *

* before. The initial decline preceded antihypertensive therapy. *
* Similarly, the decline in coronary disease mortality preceded *
* vigorous blood pressure treatment programs. Hypertension control *
* has improved in the last decade.However, this does not complete- *
* ly explain observed decreases in Black CVD mortality rates. $\quad \stackrel{+}{*}$
* 

If hypertension exerts a major force on mortality in the Black population, do recent trends in hypertension awareness, treatment, and control account for reductions in hypertension-related mortality? The analysis by Hardy and Hawkins(114) of the impact of antihypertensive therapy on mortality among mild hypertensives in the Hypertension Detection and Follow-up Program reveals that $36 \%$ of the overall mortality reduction is attributable to indices of treatment, measured annually. It is clear from examination of the referred care group in this study that hypertension control was not as vigorous in the community as in the stepped care intervention group, and that Black subjects were less likely to be treated than whites. (115) The drug regimen to which the stepped care group was subjected was only
part of a medical care program in which the care provided for hypertension was comprehensive, of high quality, free, convenient, and easily accessible. In the fifth year of the trial, $75-82 \%$ of the subjects in the four race-sex, stepped care groups were receiving drug therapy, and only $5.2 \%$ of all the stepped care participants were lost to follow-up. Though some investigators are wont to emphasize that pharmacologic therapy is the major cause of the observed differences, to focus exclusively on the drugs is to neglect the other essential components of the health care system devised for the trial, including the "strong support system" (116) which made excellent compliance and improved noncardiovascular health outcomes(117) more likely.

As Wing has clarified $(118,119)$, consideration of hypertension-related mortality declines and improvements in hypertension treatment on an age-race-sex-specific basis indicates trends that are not consistent with the hypothesis that the mortality decline is entirely attributable to improved awareness, treatment, and blood pressure control. It is probably inappropriate to regard improved hypertension control in Blacks as a complete explanation for trends in hypertension-related mortality.

## B: Socioeconomic Factors



* There are persistent differences between Blacks and whites in $\Rightarrow$
* education, occupation, and income. On average, Blacks have less *
* education than whites. Those with equivalent education have $\Rightarrow$
$\Rightarrow$ access to fewer job opportunities than whites. Those with equiv- *
$\stackrel{\Delta}{\circ} \quad$ alent employment are likely to be paid less than whites. $\quad *$



## Social Epidemiology

There is abundant evidence of disparities between Blacks and whites in the U.S. in income, occupation, and education, three of the major variables employed for assessment of socioeconomic status (120-124). Table 29 shows the ratio of non-white to white median income in the U.S. from 1945 to 1977 for men and women and lists such data specifically for Blacks from 1964.

The distribution of family income in Black and white households, categorized according to the gender of the head of the household is shown in Table 30. The distribution for Black families, especially for those headed by women, is skewed toward the lower incomes. Despite a rapid increase in the percentage of white-collar workers and rapid declines in the percentage of farm workers among non-whites, especially non-white women, in the period 1963 to 1973, the vast majority of non-whites, especially men, remain blue-collar or service workers. These changes in occupational profile occurred simultaneously with gains in the percentage of non-whites, 25-34 years old, who completed high school or four years of college. (122) By 1981, the median number of years of schooling for Black men and
women exceeded 12, and the black-white difference was less than one year. During the period 1960-1981, the median duration of formal education had increased by 4.4 years for Black men and 3.5 years for Black women, but by only 1.9 and 1.3 years respectively for white men and women. (121)

However, as a recent economic report(121) has noted: "the income gap between Blacks and whites is less related to education than to job opportunities open to Blacks." In addition, it has been shown that, for men holding the same types of jobs, Blacks commonly earn less money than whites. $(125,126)$

These socioeconomic differences between Blacks and whites are associated with important differences in the conditions of their lives and thus with their health experience. (46) The available data on the association of markers of socioeconomic status with coronary heart disease, hypertension, stroke, and end-stage renal disease are reviewed next.

## Coronary Heart Disease

> There is inconsistent evidence concerning any association bet $\Rightarrow$ ween socioeconomic status and coronary disease. There is some $\therefore$ evidence of a low incidence of coronary disease in Blacks of high socioeconomic status.
(i) Social Epidemiology

Antonovsky(127) and Jenkins(128), reviewing social precursors of CHD in white populations found conflicting evidence concerning the relationship between CHD and socioeconomic status. Kasl(129) and James (130,131) have commented on some aspects of the associations of CHD and socioeconomic status in Blacks. In Evans County, Georgia, the initial prevalence study revealed a lower prevalence of CHD among low socioeconomic status white men and the seven-and-a-quarter-year incidence data did not reveal significant differences in CHD rates between high and low socioeconomic status white men. (132) The Black men (low-SES sharecroppers) had a CHD incidence rate lower than that of white men. Tyroler et al(14) have shown that 20 -year all-cause mortality rates for Blacks and low social status whites in Evans County, Georgia, were almost identical, both being less favorable than that of higher social status white men. For white men the age-time-adjusted CHD mortality rates were higher among those of low social status than those of high social status. A similar relationship of social status to CHD incidence has been observed in Black men in the Charleston Heart Study. Keil et al(15) have reported the age-adjusted CHD incidence in high social status Black men to be 61 per 1,000 , compared to 132 per 1,000 for the randomly selected Black men who were predominantly of low social class. A report from the Beta-Blocker Heart Attack Trial (BHAT) (133) has shown that two characteristics, being socially isolated and having a high
degree of life stress, were strong predictors of 3-year mortality after myocardial infarction. Markers of a high stress level included being in a relatively low-status job (especially one that was not enjoyed) before infarction, being retired but preferring to be working, and having experienced major financial difficulty in the previous year. High levels of stress were most prevalent among the least educated, a group in which Blacks are over-represented.

## Socioeconomic Status and Biological Risk Factors

## \%

* Some data indicate associations of adverse risk factor distrib- *
* utions with lower socioeconomic status. However, there is some * * suggestion that higher socioeconomic status may be associated * * with lower levels of HDL-cholesterol. Social mobility would also * * be expected to play a role, with socioeconomic status, in risk *

One epidemiologic study has revealed an inverse relationship between socioeconomic status and the prevalence or severity of some CHD risk factors, but this applies to all the ethnic groups examined except Blacks. (134) In NHANES I, examination of behavioral and demographic variables related to serum cholesterol revealed that serum cholesterol levels were significantly higher in the lowest socioeconomic class (scored by educational attainment and income levels) for white people, but not for Blacks. (54) Overall mean serum cholesterol levels for Black men, ages 18-74 years, showed no significant changes as income levels increased. In contrast, among Black women the mean serum cholesterol levels in those aged 18-74 years generally decreased as income increased and, across the income levels, Black women had generally lower mean serum cholesterol levels than did white women. There is a suggestion from the Framingham Minority Study (135) that Blacks of higher socioeconomic status might have lower HDL-cholesterol levels than those observed in the general Black population.

In a Cincinnati study (136) there was an inverse relationship between socioeconomic status (assessed by education and occupation of the head of household) and smoking in both children and adults.

A significant inverse association was detected between relative weight and duration of formal education in the $\operatorname{HDFP}(137)$. The data on the associations between socioeconomic status and hypertension in Blacks are reviewed in the next section.
(i) Social Epidemiology

There is an inverse association of education with hypertension * prevalence which is common to both Blacks and whites. Some stud- * ies of Blacks have detected inverse associations of blood * pressure prevalence and/or incidence with both income and social * class.

For both Blacks and whites, a distinct social class/blood pressure gradient exists with those of lower income and lower educational attainment having higher blood pressures. In the HDFP screening(136), there was an inverse relationship between blood pressure (and the prevalence of hypertension) and the number of years of education [Figure 15]. This relationship was more striking for Blacks than for whites and persisted even after account was taken of body weight. For example, in Blacks with less than 10 years of education, the prevalence of hypertension was $43.9 \%$, but in Blacks who had completed college education, the prevalence was $27.7 \%$. The corresponding rates for whites were $23.1 \%$ and $13.5 \%$. The persistent Black excess of hypertension across all educational levels has been reported in national health surveys and mean systolic and diastolic blood pressures were inversely related to the amount of formal schooling received by examinees in all race and sex groups in NHANES II. This association was more marked for women than for men. A similar association of per capita income to blood pressure level has been observed in a 1981 survey of individuals examined in a representative random sample of Georgia households. (138) Per capita income was significantly lower among individuals with moderate or severe hypertension than in those whose hypertension was mild or controlled [Table 31]. This applied to all race-sex groups except white women. In studies that employed social class categories (assessed by education and occupation), a higher prevalence(139) and incidence (140) of hypertension has been detected in Blacks of low social class. In the latter investigation, a community-based study in Charleston County, (140) the association of hypertension with skin color was minimal and substantially less than the association with social class. These investigators concluded that social class may be among the primary determinants of hypertension in Blacks. Similar observations have been made in a study of the incidence of hypertension in an inner-city (Baltimore) Black population(107): in both sexes there was an inverse association between the incidence of hypertension and income. Sons of professionals had an incidence of hypertension, over a three- to four-year period, that was approximately one quarter that of sons of laborers.

## -

* The mechanism by which socioeconomic status is associated with * high blood pressure in Blacks is unclear. The data show some * high social stress and instability, and interactive influences * of coping styles, education, and occupational insecurity on
of hypertension. $\quad *$

为
The studies cited do not go beyond a demonstration that an inverse association exists between socioeconomic status and the risk of hypertension in Blacks. It has not been satisfactorily determined how social and economic factors contribute to this association, nor what the factors are that lead to individual variation. Harburg et al(141-143) have sought to clarify some of these ideas. They formulated a hypothesis that populations in different urban areas with varying rates of stressful conditions might manifest variations in blood pressure levels, and sought to test. this hypothesis by comparing urban areas in which social life was disorganized with those characterized by greater social organization. The areas of social disorganization were characterized by greater unemployment, lower per capita income, lower percentage of home ownership by occupants, higher juvenile delinquency rates, and higher adult crime rates against people and property. Such areas were described as "high-stress" areas and the areas of greater social organization were regarded as "low-stress" areas. These investigators observed that Black men in "high-stress" areas had higher age- and weight-adjusted systolic and diastolic blood pressures than Black men in "low-stress" areas, but similar relationships did not hold for Black women or white men. In particular, this effect appeared to be confined to men less than 40 years old: young Black men in "high-stress" areas were 2.5 times as likely to have diastolic blood pressures greater than or equal to 90 mm Hg than those living in "low-stress" areas.

In commenting on these observations, Tyroler and James (144) suggested that such chronic blood pressure elevations might be related in part to struggles by younger Black men to acquire the economic and social resources to control their environment in circumstances largely beyond their control. James et al(145,146) have recently presented data concordant with certain aspects of this hypothesis. Observations on a sample of young Black men in rural North Carolina suggest that the stance of coping actively with a difficult environment is associated with higher blood pressure if the individual lacks the educational or other resources which would allow effective coping. Job insecurity was associated with higher blood
pressures, and a man's perception that being Black had hindered his chances for achieving job success was also associated with higher blood pressures in those men with an active coping style.

The National Heart, Lung, and Blood Institute has recognized the need for further elucidation of the interactions between social and psychological factors and the risk of hypertension and has sponsored a series of investigations of "Biobehavioral Factors Affecting Hypertension in Blacks."

## (iii) Social Class and Hypertension-Related Mortality

## 

* Hypertension-associated mortality rates also show associations * with social instability.

As would be expected from the inverse association between social class and the prevalence of hypertension, there is a similar gradient between mortality related to hypertensive disease and social class in the Black population.(147) Examinations of stroke mortality in the counties of North Carolina in the early 1960s revealed higher rates in Black populations characterized by greater familial and social disorganization [Figure 16](148). A subsequent examination by James and Kleinbaum(149), with variables similar to those used by Harburg et al, demonstrated that hypertension-related mortality rates were more closely associated with social instability than with socioeconomic status [Figure 17]. On the individual rather than the ecologic level, Tyroler(75) has demonstrated an inverse relationship between education and five-year, age-adjusted mortality in white men, ages 40-69 years, in the referred care group of the Hypertension Detection and Follow-up Program with entry diastolic blood pressure, 90-104 mm Hg. The Black men with less-than-high-school education had an age-adjusted mortality rate 1.4 times that of white men of similar education, 2.3 times that of white men who had completed high school, and 3.8 times that of white men who had more than a high-school education [Table 32]. Part of this race difference reflects differential treatment rates between Black and white men in the referred care group. (115)

Stroke
(i) Links with Socioeconomic Factors

The analysis by James and Kleinbaum(149) suggested that the ecologic association between stroke mortality rates and familial and social disorganization in Blacks in North Carolina counties was related more strongly to social instability than to low socioeconomic status.
(i) Links with Socioeconomic Factors

In view of the inverse association between hypertension prevalence and severity and socioeconomic status, it is likely that incidence rates of hypertensive end-stage renal disease are higher in
low-income Blacks. This may be related to inadequate treatment of hypertension, particularly in those with the lowest incomes. The Georgia 1981 survey (138) revealed that those with the most severe hypertension, in addition to being the poorest, would have to spend the largest proportion of their income on medications to attain adequate blood pressure control [Table 33].

## C: Behavioral and/or Cultural Factors

## Introduction

Some of the factors that may increase risk of cardiovascular disease, such as cigarette smoking and physical inactivity, are behaviors. Other physiological characteristics that may enhance risk are themselves the consequence of dietary and other behaviors. Some of these behaviors are part of particular cultural patterns, many of them grounded in socioeconomic circumstances associated with increased risk. In addition, certain cultural patterns may impede efforts to reduce risk. In particular, cultural factors may influence the effectiveness of efforts to prevent hypertension, to reduce CHD risk by reducing risk factors, and to treat hypertension more effectively. This section reviews evidence from national surveys and other studies on health beliefs, health practices, and health-relevant behavior that have implications for cardiovascular disease in Blacks.

## Coronary Heart Disease

(i) Health Beliefs/Knowledge

## ※

* Data on Blacks' beliefs and knowledge of coronary heart disease * * are inadequate. Available data suggest significant deficits in * * Blacks' knowledge concerning the association of CVD with diet. *


Some recent data on health beliefs, knowledge, and information in Blacks concerning coronary heart disease and on health practices which predispose to it are available. Such data appear to be less ample than the data for hypertension.

In 1982, Gillum and Grant(48) could identify only one study which assessed awareness of CHD risk factors in the Black community. This study examined a random sample of 300 adults, ages $18-65$ years, in three predominantly Black neighborhoods in a southern metropolitan city. (150) Subjects were appraised for their competency in recognition of CHD, its signs and symptoms, as well as for their basic knowledge of habits or lifestyles that increase the risk of CHD. The data revealed a low level of awareness of CHD risk factors among the sample population. The level of knowledge was correlated with age, educational level, and socioeconomic status.

In a 1979 Louis Harris - Urban Behavioral Research Associate Survey (151), Blacks were somewhat less likely than whites to identify obesity ( $24 \%$ vs $36 \%$ ), cigarette smoking ( $20 \%$ vs $34 \%$ ), lack of exercise ( $11 \%$ vs $22 \%$ ), fatty foods and cholesterol ( $5 \%$ vs $11 \%$ ), as likely causes of heart trouble. They were equally likely to identify high blood pressure ( $24 \%$ vs $25 \%$ ), excessive alcohol consumption ( $10 \%$ vs 9\%), and emotional pressure, worry, and anxiety ( $39 \%$ vs $40 \%$ ) as causes of heart trouble. Blacks were also less likely than whites to identify proper diet ( $38 \%$ vs $55 \%$ ), exercise and staying in shape ( $27 \%$ vs $48 \%$ ), and smoking cessation ( $9 \%$ vs $22 \%$ ), as the best ways of preventing heart trouble.

## (b) Diet and Cardiovascular Disease

| Data on Blacks' health practices important for coronary disease outcomes are scanty. Few demonstration and education research efforts have sought to apply insights obtained from studies of |
| :---: |
|  |  |
|  |  |
|  |  |

A 1982 telephnne survey(152) of a national probability sample of 1,000 subjects, performed by the FDA and the NHLBI, assessed beliefs and knowledge concerning the relationships between diet and cardiovascular disease. The sample included 9\% of Black subjects. Examination of the Black subset revealed that Blacks showed less-than-average awareness of diet-health relationships [Table 34]. In particular, there was relative unawareness of the association between diet, especially fats and cholesterol, and cardiovascular diseases other than hypertension. Analysis showed that the undereducated, low-income respondents and those living in the South were also likely to have less-than-average awareness of diet-health relationships. Educational level was positively correlated with concern about consumption of fats and cholesterol. After adjustment for educational level, the concern Blacks had about possible adverse effects of consumption of fats and cholesterol was actually greater than the average. However, in view of the educational disadvantages of Blacks discussed earlier, this statistical adjustment would obscure a need for greater cardiovascular health education efforts, especially among poorly educated Blacks.

The impact of formal education, especially of reading achievement, has been emphasized in some studies of cardiovascular health education. For example, in one school-based, cardiovascular health education study in Chicago, Sunseri et al(153) detected racial differences in the increases in knowledge concerning nutrition, exercise, and smoking and their relationship to cardiovascular disease after an intervention. Black children had a smaller increase in knowledge than others and, at follow-up nine months later, had persistently lower knowledge scores even after adjustment for reading achievement. Black reading achievement was significantly lower than that of whites. The analyses also revealed that reading achievement was significantly related to nutrition knowledge and attitudes, but not to behavior.

## (ii) Health Practices

## (a) Dietary Practices

Some of the available data suggest differences in health practices between Blacks and whites that may be important for CHD outcomes. For example, some anthropologic data indicate that frying of foods is a very common method of food preparation among Blacks. (154) In one New York ghetto Black population, one-third of Black mothers cited frying as the method of food preparation of first choice. (155)

There are few data on the likelihood of dietary change in Blacks in response to physician advice, though in the FDA-NHLBI national survey on diet and cardiovascular disease, Blacks appeared to be more likely to be on a medically-prescribed diet that included fat and cholesterol reduction [Table 34]. In the Multiple Risk Factor Intervention Trial(156), Black men at baseline reported lower daily caloric consumption, similar consumption of saturated and of polyunsaturated fatty acids and significantly higher consumption of cholesterol than did whites. In the Special Intervention group, Black-white differences in changes in intake of specific nutrients and in weight were small, which suggests that under medical supervision, similar changes are possible in Black and white men. However, this group may not be representative of the general population.

## (b) Physical Activity

No national survey has compared physical activity patterns in Black and white populations. Thus, it is unknown whether the preponderance of Blacks in lower-status, often more physical, occupations results in higher levels of overall physical activity than in whites.

Cigarette smoking and trends in this behavior have been discussed earlier. NCHS 1978 data indicate that Black men and women, although lighter smokers, were more likely to smoke cigarettes with high tar and nicotine content than were whites. In addition, though more Blacks had tried to stop smoking at some time, fewer of them were successful. These observations are consistent with those in the Multiple Risk Factor Intervention Trial.(156) Though Black smokers reported smoking significantly fewer cigarettes per day, Black men were less likely than whites to stop smoking and, among those who stopped, the recidivism rate was higher than in whites. The NCHS 1978 data also show that a larger proportion of Black women than men reported attempting to stop smoking, $44.2 \%$ vs $34.1 \%$.

Determinants of smoking behavior in Blacks have not been studied thoroughly. The relationship between the frequency of preventive practices and socioeconomic status appears to apply to smoking cessation in Blacks.(157) For example, there is some evidence, from examination of hospitalized controls in an American Health Foundation study(158), that education may be related to differential rates of change in the prevalence of smoking among Black men. In 1971-75, $54.2 \%$ of Black, college-educated men smoked: this percentage had declined to $36.5 \%$ by 1976-1980. In contrast, among Black men with less than high-school education, the decline was much smaller, from $59.4 \%$ to $55.1 \%$.

In addition, Black smokers who earn more than $\$ 15,000$ per year and those who report regular medical use of care are more interested in quitting smoking and report enjoying smoking less than do those with lower income and health care access.(159) The class effect may be mediated partly by absent or less frequent exposure to preventive health care services. Black smokers, especially those of low or lower middle class status, outside the traditional medical service structures are less likely to be exposed to information provided by medical care personnel on the risks of smoking and the benefits of cessation.

There is some evidence that Blacks smoke cigarettes with higher nicotine content than whites, which would result in greater physiologic dependence and would enhance the difficulty of smoking cessation. A plausible inference from the higher smoking rates and the lower cessation rates among Black smokers is that they may experience weaker social support for cessation from their primary peer and family groups and from modelling by former smokers among their peers. Smoking cessation rates among Blacks may be enhanced if their access to effective influences for smoking cessation is ensured. This may require efforts to curb the vigorous tobacco advertising directed to the Black community and delivery of preventive health services by methods outside the traditional medical care system, perhaps by approaches which employ Black community resources. Research in this area is currently being sponsored by the National Cancer Institute, and includes assessment of techniques for ensuring smoking cessation in women, and assessment of the impact of
self-help strategies, media-based instruction, and advice by physicians and dentists.

The initiation of cigarette smoking among grade-school students has been studied, and some data suggest that, after health education programs, Black students are somewhat less likely than whites to begin smoking. Data from the Bogalusa Heart Study (160) indicate that Black children smoked less and experimented with smoking later than did white children. However, they were less likely to become habitual smokers if they experimented with cigarettes before age 12 years.

## (iii) Health Care Seeking Behavior

The available data are inadequate to allow assessment of whether knowledge of the symptoms of heart disease in Blacks is comparable to that in whites. Differences in knowledge and awareness of these symptoms and in behavior in response to them could underlie differences in sudden cardiac death rates between Blacks and whites.

## Hypertension

(i) Health Beliefs/Knowledge


* Awareness of high blood pressure has increased in Blacks in the *
* last decade, but many Blacks have significant misconceptions con-* * cerning factors that predispose to hypertension. *



## (a) Hypertension as a Health Problem

The increased rates of awareness, treatment, and control of hypertension in Blacks, since 1960, revealed by the NHANES II data [Table 9](26) indicate greater knowledge of this health problem in the Black population. This is probably largely attributable to the National High Blood Pressure Education Program. Of those diseases considered most serious by the public, hypertension is the only one for which there is a substantial difference between the races in how seriously it is perceived. (151) Blacks were more likely to regard hypertension as a very serious problem than were whites ( $82 \%$ vs $72 \%$ ). This also applied to hypertensive subjects ( $89 \%$ vs $81 \%$ ). Interviewees reported that their doctor or the clinic at which they obtained medical care was the major source of health information (Blacks $83 \%$, whites $84 \%$ ). Public service messages on television were also highly rated (Blacks $66 \%$, whites $63 \%$ ), but health articles or medical columns in magazines and newspapers were rated less highly by Blacks and whites.

Awareness of hypertension as a serious health problem is not necessarily accompanied by accurate perceptions concerning the disease, its causes, diagnosis, and treatment. This has been concluded from urban(161) and rural(162) surveys as well as national
surveys.(151) Blacks are less likely than whites to know what blood pressure would be normal for someone of their age ( $18 \%$ vs $33 \%$ ). This difference was also detected in hypertensive subjects ( $22 \%$ vs $45 \%$ ), and may be related to the likelihood that Blacks are more likely than whites ( $65 \%$ vs $47 \%$ ) to be informed about their blood pressure by health care providers by means of general, descriptive phrases rather than by being told the actual blood pressure reading together with comment and/or advice.

## (b) Causes of Hypertension

In specifying likely causes of hypertension, Blacks were more likely than whites to emphasize dietary causes such as excessive salt intake ( $24 \%$ vs $11 \%$ ), intake of fatty foods or cholesterol ( $9 \%$ vs $6 \%$ ), and intake of pork ( $17 \%$ vs $1 \%$ ). It is of particular interest that, though Blacks were more likely to identify improper diet and overeating ( $36 \%$ vs $30 \%$ ) as a likely cause of hypertension, only $17 \%$ (compared to $27 \%$ ) considered being overweight as a likely cause of high blood pressure. Blacks were also less likely to regard emotional pressure, worry, or anxiety as likely causes of high blood pressure ( $38 \%$ vs $52 \%$ ). A higher percentage of Blacks had been told to decrease sodium intake than whites, but reported salt use by Blacks was not substantially different from that of whites. In the 1982 FDA-NHLBI survey (152), though more than half of the respondents were aware of the suspected relationship between sodium and hypertension, Blacks were relatively unaware of the association between alcohol and hypertension [Table 34].

Overall, comparison of the 1979 data with those from an earlier survey in 1973 indicate a sizeable increase in awareness of hypertension and of its consequences in this period. It is likely that educational programs have played a significant role in increasing public awareness and understanding.

## (ii) Health Practices

## (a) Dietary Practices and Primary Prevention

The available data permit speculation on the impact of certain health practices on hypertension in Blacks. NHANES II dietary consumption data(98), derived from dietary recall, indicate that potassium intake is lower in Blacks than in whites and that sodium intake is similar in Blacks and whites. The higher sodium-to-potassium ratio that results from this may have implications for the higher prevalence of hypertension in Blacks. It may account for part of the racial gradient by socioeconomic status, since foods rich in potassium tend to be somewhat more expensive and may, for that reason, be consumed less frequently, especially by low-income Blacks [Figure 18].(99)

NHANES II data(58) also show higher Quetelet indices, greater maximum weights, greater minimum weights, and greater weight at age 25 years in Black women than in white women. Caloric intake in age-groups 21 to 45 years, 46 to 65 years, and greater than 65 years is lower in Black than in white women and slightly but not
significantly higher in the age-group 12 to 20 years. The association between weight gain and the incidence of hypertension in Black women in Evans County (106) and Baltimore(107) has already been noted.

The National Heart, Lung, and Blood Institute is sponsoring research on the development of obesity in young Black and white women in order to determine whether ethnic differences are due to differences in psychosocial, socioeconomic, and other environmental factors. It is hoped that this will permit assessment of the influence of the development of obesity on changes in blood pressure and serum lipids, and that it might contribute to information for the design of programs for prevention of obesity in young Black and white women.

## (iii) Health Care Seeking Behavior

 $\therefore$

* Nonadherence to antihypertensive therapy is not a problem spec- *
* ific to Blacks. Determinants of nonadherence by Black hyperten- *
* sive patients have been identified in a number of studies. The * effectiveness of a number of health-education strategies in * enhancing adherence has been illustrated in studies of Black patient groups.
*
$\qquad$
$\rightleftharpoons \quad$ patien



## (a) Nonadherence to Antihypertensive Therapy

NHANES II data (1976-1980, (26)) reveal that, of those people, ages 25-74 years, with hypertension, Black men and women are slightly more likely to be on medication than white men and women, though they are slightly less likely to have their blood pressure adequately controlled [Table 9]. In the 1979 Louis Harris - Urban Behavioral Research Associates Survey(151), Black hypertensive subjects were more likely to have taken some medicine for high blood pressure (88\%) than whites ( $80 \%$ ). Of those who had taken medicines, Blacks were more likely to be still taking the medicine ( $85 \%$ vs $80 \%$ ), though of those still taking medicines, Blacks were less likely to be taking them regularly as prescribed ( $89 \%$ vs $95 \%$ ). These findings on the similarity of rates of adherence to antihypertensive therapy among Blacks and whites are in accord with Kasl's conclusion from the literature that race is not a reliable predictor of noncompliance. (163) In fact, Kasl, noting the similarity of the "superficial" demographic characteristics (young, male, Black, somewhat lower socioeconomic status) of aware, untreated hypertensives and unaware hypertensives has inferred that the successive barriers to recognition of hypertensive status and to treatment may be comparable. The NHLBI Working Group on Noncompliance in Black Male Hypertensives convened in 1982(164), though noting the need for study of health beliefs and attitudes in this subgroup, has also identified the need for structured interventions in order to increase access to the health care system for this subset of patients.(165)
\%

* A number of studies, some of predominantly Black groups of *
* patients and others of somewhat evenly mixed Black and white *
* groups, have examined determinants of adherence to antihyperten- *
* sive therapy. The results of these studies have important implic-\%
* ations for hypertension control in Black populations.

Caldwell(166), in a pilot study of social and emotional factors influencing a patient's ability to follow antihypertensive therapy, compared a group of dropouts from antihypertensive therapy who later developed a hypertensive emergency, with a radically different group of patients who had remained in treatment for more than five years. The dropouts were more. likely to be non-white, less-educated, of lower occupational status, to have lower incomes, to be younger, and to have a briefer duration of disease. In contrast, Nelson et al(167), in a study of 142 hypertensive patients attending a medical clinic at a large urban hospital, detected no independent relationship between race or socioeconomic status and compliance with antihypertensive therapy. Characteristics of those patients less likely to be compliant were male gender, social isolation, and the presence of side effects of antihypertensive medications. In this study, the impact of side effects on compliance appeared to be greater in Blacks than in whites. However, in the 1979 National Survey (151), 18\% of Blacks and $17 \%$ of whites compliant with medications reported side effects and, among noncompliers, whites were much more likely to say that the medicine had side effects. Two analyses of patient participation and adherence to therapy have been published from the Hypertension Detection and Follow-up Program. $(168,169)$ At the end of the first year, Black men and women were less likely to be in active treatment ( $76.6 \%, 78.6 \%$, respectively) than white men and women ( $87.1 \%$, 81.9\%). Employment status in Black men under 50 years was a predictor of active status: these men were more likely to be active at the end of the first year if employed full-time than if they were not so employed. Similarly, Black men under 60 years of age were more likely to be in active treatment if they had more than high-school education than if they were not high-school graduates. Thus, markers of socioeconomic disadvantage were associated with lower rates of adherence to therapy. A subsequent analysis that considered four-month periods within the first two years of follow-up found that being Black was a predictor both of passage from active to inactive status (though not significant at the $5 \%$ level) and of passage from inactive to active status.

Other investigations of predominantly Black patient groups have revealed a variety of predictors of compliance and noncompliance. In 1979, Hershey et al(170) examined a 92.5\% Black sample, $56 \%$ unemployed, greater than $50 \%$ with a family income of less than $\$ 5,000$. They found that perception of high control over health matters, a minimum of unfavorable attitudes to antihypertensive
medications and a short duration of therapy were all associated with compliance with therapy. Cummings et al(171), in a study of 206 patients, $97 \%$ Black, with a median family income in 1978 of less than $\$ 4000$, found that sex, age, health beliefs, and experience of side effects were not major determinants of poor compliance. People who perceived their health status as poorer than others and who had a history of heart trouble were more likely to remain in therapy. Dropouts actually reported fewer problems with access to care, and more of them had medical insurance.

## (c) Effective Intervention Models

※

* Hypertension control in Black communities can be improved by *
* interventions that are not strictly biomedical and, instead, *
* either assist patients in the fulfillment of their social needs *
* or increase levels of social support.
* 



Kasl(172), in articulating a social-psychological perspective on successful community control of high blood pressure, arrived at the following conclusion: "what is apparently needed are activities which represent a good deal of contact with and monitoring of the patient at his or her convenience, usually in the patient's home, with some possible spillover into general social work activities to help the patient with his social needs." Such care, he avers, produces better blood pressure control above and beyond compliance with medications.

Syme(173), reporting on a study based in a neighborhood clinic serving a low-income community with $85 \%$ Black patients, $90 \%$ of them medically indigent, described the differential impact of three approaches to therapy. One was the "medical" approach in which the clinic physician gave care in the usual manner. The second was a "group" approach in which patients attended 12 weekly meetings with a health educator and a nurse-practitioner in which the intention was to teach them about hypertension and its management. The third was an "outreach" approach in which patients were visited in their homes by a community health worker who had been trained to help the patients meet their diverse medical and social needs. After a seven-month interval, the "outreach" group had significantly more patients under control than either of the other groups, and compliant patients in the "outreach" group were twice as successful in attaining control as compliant patients in the "group" approach. This study suggests that: "hypertension control may be achieved, at least in part, by reduction of specific types of stressful situations (family difficulties, financial hardships, employment problems) and by assisting people to make appropriate adaptive responses." It should be noted that these data on the additional impact of psychosocial intervention on blood pressure control are consistent with a report by Caldwell et al(174) of the adverse effect of
unfavorable psychosocial and socioeconomic circumstances on blood pressure control in a Detroit population of both Black and white patients.

The impact on Black hypertensive patients of social support from family members or a peer group has been demonstrated by investigators at Johns Hopkins University. (175-179) They presented data on a controlled educational trial, in a Black inner-city clinic patient population, that assessed the impact of three interventions, singly and in combination: an exit interview to review the drugs and to increase the patient's understanding of the medical regimen; a home-visit to enhance family support for drug and dietary therapeutic measures; and a small group intervention designed to enhance the capacity of subjects to deal with their blood pressure problem. Significant differences were observed between the control group and the intervention groups in blood pressure control and hypertension-related mortality at five years. In particular, in this group of predominantly Black, inner-city, hypertensive patients, the family support and small group interventions were the most effective.

Whitehead et al(180) have described an intervention employed in a poor, rural, Black community in Mississippi(181), that was based on home-visitation by a specially trained, hypertension health counselor. These counselors were also responsible for training and monitoring volunteer counselors who became leaders of self-help groups either within extended families or in church settings. The investigators were able to compare improvements in the proportions of subjects with controlled hypertension among single clients of the hypertension health counselor and in the two types of self-help groups, the extended family and church groups. They observed that, after six months, a significantly greater proportion of hypertensives was controlled in the extended family setting than in the church groups or among the clients of hypertension health counselors.

## D: Access to and Utilization of the Health Care System

## Introduction

Because the chronic cardiovascular diseases require ongoing contact with the health care delivery system for their prevention and/or treatment, they pose special problems with regard to access to and utilization of the health care delivery system, and with regard to coordination and continuity of this process.(182) This is especially so for disadvantaged population groups, among whom Blacks are over-represented. In the last two decades, several federal programs have been established to improve access to health care for the disadvantaged(183-185). The evidence suggests that, though such programs have decreased disparities in access to care and have increased utilization rates, substantial problems with the adequacy of care remain. (184-188) Measures of access to and utilization of health services, though largely accounting for differences between majority and minority health status $10-15$ years ago, may no longer be
sufficiently sensitive as proxy measures of the adequacy and appropriateness of care received. Studies from the Indian Health Service, though describing a different minority population, suggest that increasing access to and utilization of health services does not necessarily result in a coordinated and continuous process of care. $(189,190)$ There is some evidence that subtle changes in the structure and organization of services designed to increase access may militate against coordination of care for that subset of the population at highest risk. $(191,192)$ Still, most available data comparing health care for majority and minority populations continue to emphasize access. Although the data suggest a continuing disparity in access to care, they may understate the magnitude of the disparity in terms of the adequacy of care actually received. Adequate treatment of underprivileged or disadvantaged population groups places special demands on the health care delivery system. In this respect, the chronic cardiovascular diseases, hypertension and CHD, differ significantly from each other. Care-seeking and diagnosis may not be initiated by the patient, since hypertension is generally asymptomatic. Once the disease is diagnosed by community screening or by "routine" examination, however, the patient must be made to recognize its importance if long-term treatment is to be undertaken. In addition, the necessity for continuous treatment requires that health care be obtainable in a convenient location, that costs of visits to providers and of drugs be affordable, and that all aspects of patient-provider interaction be characterized by awareness of and sensitivity to the patient's life situation. In contrast, in the case of CHD, symptoms (usually chest pain) provoke health care seeking. Misinterpretation of symptoms by the patient may delay health care seeking, and misinterpretation by the provider may lead to inadequate or inappropriate care. For those who seek care and who do not die suddenly, myocardial infarction marks the onset of a different kind of interaction with the health care system in which risk factor modification becomes necessary. In addition, more expensive diagnostic and therapeutic options such as coronary arteriography and coronary revascularization surgery, components of good quality care for significant subsets of patients with CHD, may become necessary.

In discussions of access to care, especially as relevant to chronic cardiovascular diseases, the multidimensional framework developed by Penchansky and Thomas(193) allows examination of a number of important aspects of the health care delivery system. These authors have described four medical care access dimensions which are important in this context:

1. Accessibility or location of health services in relation to the location of the clients,
2. Accommodation, or the relative ease in getting appointments with providers,
3. Affordability, or the cost of medical care and perceived worth of care in relationship to cost, and
4. Acceptability, or how well clients get along with their providers and the providers' support staff.
Related to these issues of the availability and obtainability of care and patient satisfaction with care are others largely determined by structural aspects of the health care system, such as comprehensiveness, continuity, and coordination of care.(182) These latter have significant consequences for the quality of medical care, considered from the point of view of both process and outcome.

## Health Care for Coronary Heart Disease

(i) Data on Office Visits
\#W,
$\%$ Blacks make fewer office visits for coronary disease than do $\%$ * whites, and are less likely to be seen by CVD specialists. This * * may contribute to less frequent diagnosis of coronary disease in * * Blacks.

Data from the National Ambulatory Medical Care Survey, 1975-76(194), on office visits for diseases of the circulatory system indicate an average annual rate of office visits for coronary heart disease (ICDA 410-413) in non-whites which is a little over half of the rate for whites, $42 / 1000$ compared to $80 / 1000$ population [Figure 19]. Ninety-four percent of visits for acute ischemic heart disease (ICDA 410-414) were paid by whites, and only $6 \%$ by non-whites. Similarly, 92-93\% of visits for chronic ischemic heart disease (ICDA 412) and angina pectoris (ICDA 413) were paid by whites [Table 35]. It should be noted that these data may reflect disparities in prevalence of CHD and in interpretation of symptoms by patients, but since the visit data are reports of diagnoses made by physicians, they could also reflect less frequent diagnosis of coronary disease in Blacks. Perhaps an indication of this is the fact that Blacks were somewhat less likely to have an ECG performed (2.7\%, compared to $3.3 \%$ for patients of all races). The median visit age for acute ischemic heart disease in whites was 63.2 years, while in non-whites it was 51.3 years, which suggests that the onset of CHD may be earlier in Blacks. However, the latter estimate is somewhat unreliable on account of the small sample size.

In the National Ambulatory Medical Care Survey (1975-1976), office visits of all kinds by Black patients were most likely to be to general and family practitioners ( $46.6 \%$ ) : $10.7 \%$ of visits were to internists and $0.8 \%$ to cardiovascular disease specialists. In contrast to the latter, $1.2 \%$ of visits by all patients were to cardiovascular disease specialists. Thus, Black patients are less likely than white patients to see cardiovascular specialists. This
may reflect referral patterns and diagnostic habits of physicians and probably has an adverse effect on the exposure of Black patients with CHD to accurate diagnosis and appropriate treatment.

## (ii) Hospitalization

A 1982 U.S. survey(188) confirms that, in traditionally disadvantaged groups, including minorities, the unemployed and the poor are highly unlikely to obtain medical help when they need it. In this survey, $2 \%$ of American families had experienced a serious illness that caused major financial problems during the previous year. Six percent of families reported that they needed medical help during the year but failed to get it, and $2 \%$ of families were refused care for financial reasons. Blacks and other minorities are over-represented in these subsets.(185)

There are some indications that members of minority groups admitted to public hospital emergency rooms for evaluation are sometimes transferred to other facilities, despite the risk of life-threatening arrhythmias, because of their inability to pay for medical care. (194a) Many examples of "dumping" of poor patients for economic reasons have been provided by other observers. (194b,194c)

## (iii) Prognosis After Myocardial Infarction

In a Baltimore study(195) of Black and white patient groups assembled in 1966-67 and 1971, in-hospital case-fatality rates (both crude and adjusted for a number of prognostic variables) were not significantly different between Blacks and whites ( 21.0 vs $24.2 \%$ ). Follow-up of $94 \%$ of these patients ( $90 \%$ white, $85 \%$ Black) revealed no significant differences between 3 -year case fatality rates in Blacks and whites. In contrast, Shapiro et al(196) reported a $48 \%$ risk of dying within one month of first myocardial infarction in non-whites and 35\% among whites. Over the next $31 / 2$ years, the death rate among non-whites ( $23 \%$ ) was almost twice that noted for whites ( $12 \%$ ). Because of the small numbers, these data do not provide a definitive answer to the question of the prognosis of Blacks compared to whites after acute myocardial infarction.

In a study of 197 consecutive patients ( $10.7 \%$ Black) discharged after acute myocardial infarction from a metropolitan hospital in North Carolina, Kottke et al(197) observed that the lower social class patients (19 Black of 116 total) had poorer prognoses, perhaps because of other medical conditions, than those of upper social class. In particular, uncontrolled hypertension in lower social class patients was a significant predictor of new cardiac events.

## (iv) Hospitalization for Chronic Coronary Heart Disease

Data on hospital admission for chronic ischemic heart disease do not provide definitive evidence for a Black disadvantage. Yelin et al(198), in an examination of the 1976 National Health Interview Survey, found that a reported lack of insurance coverage resulted in fewer hospitalizations in a year for chronic ischemic heart disease
when account was taken of symptoms and demographic characteristics. This suggests that Blacks would be less likely to be hospitalized for chronic CHD. However, race was not a significant predictor of the likelihood of hospitalization. This may reflect, as the authors noted, the small number of minority patients (less than $10 \%$ ) in each of the samples with chronic ischemic heart disease, rather than true equivalency of hospitalization rates. However, there is ample evidence that minority groups are over-represented among the underinsured.(185)

## (v) Coronary Arteriography and Coronary Bypass Surgery

* 
* Blacks undergo coronary arteriography less frequently than * * whites. Even when Blacks and whites have coronary disease of com-* * parable severity, Blacks are less likely to undergo coronary by- * * pass surgery. (\%

The 1979 data from the National Center for Health Statistics indicate that the rate of cardiac catheterization among Blacks (1.15/1000) was only $60 \%$ of that reported for whites (1.93/1000 population).(199) Similar data also indicate that the proportion of Blacks selected for coronary artery bypass surgery among those who undergo coronary arteriography is relatively low. In 1982, only 4,000 out of 170,000 coronary bypass procedures were performed on Black patients.(200)

In a 1970-78 study in Birmingham, Alabama(201), at a medical center with a patient population approximately $1 / 3$ non-white, $96 \%$ of 6594 patients undergoing coronary arteriography were white, and only $4 \%$ Black, a ratio of $24: 1$. White patients were 2-3 times as likely to undergo coronary artery bypass surgery as Blacks even when disease severity was similar. In a study performed at Johns Hopkins University in the 1970s, only $8 \%$ of 1000 patients who underwent coronary arteriography were Black. (202) Watkins et al(203) have reviewed the clinical data on myocardial revascularization in 56 consecutive Black patients at Johns Hopkins University in the 8-year period from 1972-1980. They observed that: "the single most impressive finding in this study was the advanced state of disease in the urban Black undergoing coronary artery bypass surgery. Fifty percent of the population studied had already progressed to unstable angina by the time of the study."

Health Care for Hypertension
(i) Data on Visits to Physicians
\%

* Black patients are less likely than white patients to be seen in \% * physicians' offices, and more likely to be seen in hospital cli- * * nics or emergency rooms. For hypertensive Black patients, this * probably has adverse effects on the continuity of care received. *为

In the National Ambulatory Care Survey, 1975-76(204), hypertension accounted for $6.8 \%$ of all patient visits. This included visits at which hypertension was listed as the principal diagnosis ( $4.2 \%$ ), as well as those at which it was the second- or third-listed diagnosis. In this survey, (205) 2.1\% of visits by Black patients were for high blood pressure, compared to $1.3 \%$ for patients of all races. During office visits, Black patients were somewhat more likely to have their blood pressure checked ( $39.9 \%$, compared to $33.2 \%$ for patients of all races). In the National Ambulatory Care Survey, 1980 (206), hypertension accounted for $9 \%$ of all patient visits, and in about $89 \%$ of these visits, patients were provided with medication as therapy. Though Blacks constitute $11-12 \%$ of the United States' population and have a higher prevalence of hypertension, only $11.7 \%$ of all office visits for essential hypertension were paid by Blacks (compared to $87.7 \%$ by whites). This statistic can be understood only in the light of other information from this survey.(205) Visits to physicians in hospital clinics or emergency rooms constituted $11.2 \%$ of physician visits by whites and $25.6 \%$ of physician visits by Blacks. In addition, $69.2 \%$ of all visits by whites were to physicians' offices, compared to $58.1 \%$ of visits by Blacks. Whites were also much more likely (13.1\%) to have contact with the physician over the telephone, compared to $5.2 \%$ for Blacks.(207) Recent data also confirm that non-whites are still less likely than whites to see one particular physician for medical care: 68\% of non-whites had a regular family physician, compared to $78 \%$ of whites.(188) These data on the location of care and the ease of contact with a physician suggest that continuity of care, particularly for conditions such as hypertension, is compromised more often in Blacks than in whites.
(ii) Awareness, Treatment, and Control Status
*

* Awareness of blood pressure status and the use of antihyperten- *
* sive medications by hypertensive subjects are higher in Blacks *
* than in whites. Blacks are as likely as whites of the same sex $\%$
* to have their blood pressure adequately controlled. Black men *
* have much lower hypertension control rates than Black women. $\quad$ *
* 

These data on hypertensive patients in contact with the health care delivery system should be juxtaposed on the data cited earlier on
rates of hypertension awareness, treatment, and control [Table 9]. The NHANES II data(26) indicate that, despite recent improvements, $35.7 \%$ of Black male and $14.5 \%$ of Black female hypertensive subjects, ages 25-74 years, were unaware of their elevated blood pressure (not significantly different from $40.6 \%$ and $25.2 \%$ for white men and women, respectively). Of aware Black hypertensive subjects, $59.1 \%$ of men and $39.4 \%$ of women were not currently taking antihypertensive medications (not significantly different from $61.7 \%$ and $41.4 \%$ for white men and women, respectively). Of aware Black hypertensive subjects, $83.9 \%$ of men and $61.7 \%$ of women did not have their blood pressure adequately controlled (similar to $79.1 \%$ and $59.7 \%$ for white men and women). Though a higher percentage of hypertensive Black subjects reported taking medications, the percentage of hypertensive Blacks with adequate blood pressure control is not significantly lower than in whites, but control was significantly less likely in Black men than in Black women.

Data from selected statewide high blood pressure control programs indicate that there is substantial regional variation in the degrees of awareness, treatment, and control when Blacks and whites from the same state are compared [Table 36].
(iii) Perceptions of Access and Impact on Medical Care Use


* Some data indicate that Blacks perceive the medical care system $\%$
* to be less accessible to them. These perceptions have been chan- *
* ged and medical care use increased in a number of settings by *
* targeted interventions. Such interventions must be persistent, *
$\%$ apparently, if good results are to be preserved. $\quad \therefore$
以
A 1980 community survey in Edgecombe County, North Carolina(207), showed that, compared to whites, Blacks used the medical care system on the basis of need less frequently, had more difficulties in entering the system, and expressed greater dissatisfaction with medical care services. It is of interest that these results were not specific to hypertensive patients. Since they applied to the normotensive Black population as well, they suggest that cultural factors have an adverse impact on the appropriate use of medical care in that rural setting. An analysis of men (races combined) revealed that men aware of their hypertensive status but currently untreated had significantly more problems getting to the doctor than did those who were treated. Women who were aware but untreated were less likely than those treated to consider the health services accessible and the cost of medical care affordable.

It is in the light of such observations that the high Black drop-out rates from antihypertensive therapy in a number of urban and rural areas should be considered. The characteristics of the health care delivery system that account for this vary with the clinical setting. Some clinic-based studies have demonstrated the beneficial impact on clinic attendance and blood pressure control of introducing an appointment system which minimizes patient inconvenience and
waiting time(209), taking steps to improve the provider-patient relationship, and introducing simple, inexpensive methods of monitoring follow-up and control rates in the clinic population. The necessity for the maintenance of efforts at maximizing follow-up for indefinite periods has been illustrated by the work of Wilber and Barrow(210): an increase in the proportion of hypertensive subjects under good control from $15 \%$ to $80 \%$ in a two-year period when public-health nurses did home follow-up visits was followed by a decline to $29 \%$ two years after the program ended.
(iv) Effects of Decreasing Public Support for Health Services
*

* When care for hypertension is sustained and accessible, the im- *
$*$ pact on blood pressure control is significant.


There is convincing evidence that, when comprehensive care is offered, treatment and control rates improve and mortality rates decline. This was illustrated in the Hypertension Detection and Follow-up Program in which, as a part of that effort, the investigators provided transportation when necessary, convenient care, and free medications to stepped care participants. Recently, Brook et al(211) have provided evidence consistent with these observations in a population of about 4,000 people under age 65 years who were provided with free medical care. The improvements in blood pressure control and the reduced risk of dying from complications of hypertension brought about by free care were largest in the group with the lowest income and the highest relative risk. The results of withdrawal of such care are evident, from the study reported by Lurie et al(212) on a group of poor, chronically ill subjects, $55 \%$ Black or Hispanic, whose care under the California Medicaid program was terminated. In these patients, blood pressure control deteriorated during the six-month period following termination and this adverse outcome was sustained at one year(213), particularly among those medically indigent individuals who remained uninsured.

With regard to this, an interesting use of local public funds for blood pressure control [Table 31] has been undertaken in the State of Georgia. Antihypertensive medications are provided free to medically indigent hypertensive patients not eligible for Medicaid or other third-party reimbursement. (214)

## (v) Black Physician Manpower

The small proportion of Black physicians in the U.S. has the consequence that most ambulatory care for Black patient is provided by non-Black physicians(215) even though Black physicians are far more likely to serve Black patients than are non-Black physicians. The data show that $87 \%$ of visits to Black physicians were made by Black patients, but only $7.4 \%$ of the visits to non-Black physicians were made by Black patients. In absolute terms, however, most Black patients visit non-Black physicians. The total number of visits by

Blacks to non-Black physicians was 41.6 million and to Black physicians 5.1 million. About $59 \%$ of Black physicians were internists and general or family practitioners.

Another consequence of the relatively small number of Black physicians is that the likelihood that a Black patient would be seen by a Black cardiologist with whom there would be some cultural affinity, is very low. The best available information on the number of Black cardiologists who have Board Certification in Internal Medicine is from the records of the Association of Black cardiologists. The number of such physicians in the United States is less than 80. Lack of awareness among white physicians, by whom most Black patients are seen, of the importance of coronary disease in Blacks may have adverse impact on diagnostic and therapeutic practices.

CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN HISPANIC, ASIAN/PACIFIC ISLANDER, AND NATIVE AMERICAN POPULATIONS IN THE UNITED STATES

## Introduction

The extent and quality of the data on the impact of cardiovascular diseases in Hispanic populations is even more limited than that available for Blacks. Part of this limitation is due to the occasional practice of aggregating the mortality data on white Hispanics into a non-white category. An additional complicating factor is that few studies on cardiovascular diseases that include these populations separate the ethnic subgroups or nationalities they study. Thus, for example, studies that report results by major ethnic groups typically use a general classifier such as "Hispanic" to include such ethnically diverse groups as Mexican immigrants, native-born Mexican Americans, Puerto Ricans, Cubans, and immigrants from Central and South America. Similar problems occur with Native Americans when classifications ignore important tribal differences, and with Asian/Pacific Islanders when ethnic differences and nationality within these groups are also ignored. Therefore, not only are we relying on limited information on which to draw conclusions about the cardiovascular disease status of these populations, but the available data often does not permit us to make reliable statements about CHD morbidity and mortality rates in specific subgroups or to generalize beyond the specific groups on which the data are based (e.g. data on cardiovascular disease in immigrant metropolitan Puerto Ricans is probably of little utility in estimating CHD mortality rates in rural, native-born Mexican Americans).

In addition to these limitations in the quality, specificity, and generalizability of the available data, there are also very few studies that give adequate attention to such factors as socioeconomic status (i.e. level of education achieved, income, and occupation), socioeconomic mobility (i.e. upward or downward trends in SES across generations), migration status (i.e. place of birth and relative length of time in present geographic area), urban vs rural area of residency, and level of acculturation (i.e. degree to which minorities acquire the attitudes, beliefs, behaviors, and values of the dominant culture).

All of these factors have been identified as important predictors of health status, health knowledge and behaviors, and in the pattern of health-care utilization and quality of care received by the poor and by ethnic minorities. $(300,301,302)$

Finally, most studies and national data bases can be criticized for the small and nonrepresentative samples of ethnic minorities studied. This practice precludes reliable estimation of the trends
in cardiovascular diseases in ethnic populations, and limits our ability to generalize beyond the samples studied.

In order to ensure that the burden of coronary heart disease in each of these ethnic minority groups is given adequate attention, the available evidence on coronary heart disease (CHD), ischemic heart disease (IHD), hypertension, and stroke mortality and morbidity, risk factors, and pattern of health care utilization are reviewed for each group.

## I INTRODUCTION



* Despite limited CHD mortality data availability, soon to be rem- * * edied by the Hispanic NHANES, cardiovascular disease is a major * $\%$ cause of death in Hispanics, although the rate for non-Hispanic $\%$ * whites is somewhat higher.This lower relative rate is unexpected * $\%$ given their socioeconomic profile, pronounced rural-to-urban * * migration and high percentage of immigrants, and the prevalence * * of obesity and diabetes, particularly in women. $\quad \stackrel{y}{*}$

以

Hispanics constitute the second largest ethnic minority in the U.S. with 14.6 million identified in the 1980 Census(303) This is a very diverse group that includes Mexican nationals and Mexican Americans, most of whom reside in California and Texas; Puerto Ricans, most of whom reside in New York; Cubans and Cuban Americans, most of whom reside in Florida, and a host of Other Hispanics who have migrated from several countries in Latin America and are distributed across the country. Despite the numbers and diversity of this population, national epidemiologic data on Hispanics are very limited. The forthcoming data from the Hispanic HANES will provide health status and behavior data on the first large, nationally representative sample of Mexican Americans, Cubans, and Puerto Ricans ever obtained.

A major problem faced by investigators studying Hispanics is how to reliably identify them. As noted by Hazuda, (304) investigators studying Hispanics of Mexican origin rely on everything from a simple definition to a complex algorithm that includes surname, parental birthplace, ethnic origin of grandparents, and the person's preferred ethnic identification. In the case of Cubans and Puerto Ricans, a combination of surname and nationality/birthplace are typically used.(319) Because of the geographic distribution of Hispanics it is probably wise to assume, if not otherwise stated, that studies on Hispanics in the Southwest are based on Mexicans and Mexican Americans, those in the Northeast are based on Puerto Ricans and those in the Southeast are based on Cubans. Future studies in Los Angeles County, New York, and Miami, however, will have to consider the growing populations of Latin American immigrants into the Hispanic populations in these areas.

Mortality reports from vital statistics records are also hampered by the fact that a separate Hispanic identifier is not included or required on the death certificates in some states (e.g. California). Therefore, for example, the Los Angeles County Heart Association report on cardiovascular disease mortality determined the rate of Hispanic mortality from CHD by inference from the number of deaths that occurred in predominantly ( $75 \%$ or higher) Hispanic census tracts. $(304,305)$

To further complicate the task, it should be noted that the designation Hispanic is not a racial but an ethnocultural code. As such, individuals from diverse racial backgrounds (i.e. whites, Blacks, Indians, and mixed races), but who share a common Hispanic cultural heritage are included in this population. Therefore, genetic explanations for any observed differences between Hispanics and other groups are probably unfounded unless specific assessments of percentage of genetic admixture are considered.

## Coronary Heart Disease

## (i) Mortality

为

* National epidemiologic data on coronary disease mortality in His-*
* panics are limited to date, though the Hispanic HANES study *
* should remedy this. Regional mortality rates for Mexican Amer-
* icans in Los Angeles County and Texas are lower than in whites
* for both sexes. Limited preliminary findings suggest that the
* rate of decline in CHD mortality in Hispanics may be comparable to that in whites during the last decade.
$\star$
$\star$ * * $\star$
* 

A review of the NCHS data on excess mortality from heart disease, stroke, and atherosclerosis for 1969-1971, and from heart disease, hypertensive disease, ischemic heart disease, and cerebrovascular disease for 1979-1981 did not report any results specific to Hispanics. CHD mortality data for this population were included with those of non-Hispanic whites. Therefore, it was not possible at this time to even speculate about recent national mortality trends from cardiovascular disease in this population. Proposed plans at NCHS to obtain CHD mortality data from those states that provide an Hispanic code on death certificates will help to fill this information void.

The recent report on cardiovascular disease mortality in Los Angeles County (305) showed that major cardiovascular disease is a major cause of death for all ethnic groups including Hispanics, and accounts for nearly half of all deaths in all the ethnic groups reviewed [Table 40].(305) Results on age-adjusted mortality rates per 100,000 population for Hispanic men in LA County between 1979 and 1981, as inferred from deaths in census tracts where $75 \%$ or more of the population was Hispanic, showed that mortality from major cardiovascular disease was lower for Hispanic men than for whites and Blacks ( $441.9 / 100,000$ vs 536.6 and 558.2 respectively). The same was
true for mortality from diseases of the heart (357.8/100,000 vs 432.6 and 438.9), from ischemic heart disease (220.4/100,000 vs 274.2 and 223.9), from myocardial infarction and acute IHD (98.2/100,000 vs 235.7 and 106.9 ), from chronic IHD ( $102.2 / 100,000$ vs 138.3 and 117.0), from hypertensive disease ( $20.4 / 100.000$ vs 22.0 and 57.0 ) [Table 41]. (305)

The comparable age-adjusted mortality rates for Hispanic women in LA County were similar to those of the men. Hispanic women had mortality rates that were lower than those for white and Black women from major cardiovascular disease ( $316.7 / 100,000$ vs 335.7 and 384.4 respectively), from heart diseases (242.4/100,000 vs 245.8 and 278.0), from ischemic heart disease (148.6/100,000 vs 158.1 and 158.5), from myocardial infarction and acute IHD ( $66.8 / 100,000$ vs 70.1 and 70.9), from chronic IHD (81.8/100,000 vs 88.0 and 87.6). In the case of hypertensive disease, however, Hispanic women had a higher mortality rate than whites ( $18.6 / 100,000$ vs 15.8 ), but their rate was still significantly lower than that for Black women (40.2/100,000) [Table 42].(305) These results are similar to those reported by Schoen and Nelson(306) for California, and by Bradshaw and Fonner(307) for Texas. Both studies concurred that CVD was the leading cause of death among Hispanics, and that the mortality rate for Hispanic men was lower than the rate for white men in both states ( $82 \%$ of the white rate in California and $85 \%$ of that rate in Texas). However, CVD mortality rates in California for Hispanic women were virtually identical to those for white women, but slightly higher in Texas (7\%).

It appears from the limited mortality data available that cardiovascular disease is far from uncommon among Hispanics although their relative mortality risk from CHD and related causes appears to be lower than that of non-Hispanic whites. This lower CHD mortality rate is more apparent among Hispanic men than Hispanic women. The latter appear to be slightly more vulnerable than non-Hispanic women to hypertensive disease and strokes. In all cases, however, mortality from all major cardiovascular diseases is higher in Hispanic men than in Hispanic women.

There are very few studies of secular trends in cardiovascular disease mortality that have explored whether Hispanics also show evidence of the marked decline in CHD deaths observed in U.S. white and Black populations in the last decade. Two studies of such secular trends between 1970-1976 in Texas found that the CHD mortality decline in Hispanics to be comparable to that in whites in Bexar County, (347) but that the decline was slightly less steep for Hispanic men in the entire state.(309)

## (ii) Morbidity

Data on CHD morbidity in Puerto Rican, Cuban, Mexican American, and other Hispanics are also sparse, and due to presumed group differences in a number of parameters, such as use of the traditional medical care system, severity of illness required before an individual enters the system, and variability in the disease
classification given to a specific symptom complex, the available data are considered to be of marginal value especially for comparisons among groups.

## Stroke

(i) Mortality


#### Abstract

W. * National data on stroke mortality rates in Hispanics are lack- * * ing. Compared to whites, some preliminary regional data suggests * * lower rates in Mexican Americans, but the possibility of slightly* * higher rates among younger Puerto Ricans in New York. 


In the absence of national data, no firm conclusions can be made about stroke mortality and morbidity among Hispanic Americans. Certain regional data, some of it inferred from census tract examination, give some indications of mortality rates for certain regional Hispanic subgroups. Age-adjusted stroke mortality rates per 100,000 Hispanic men in Los Angeles County, between 1979-81, were inferred from data from census tracts where $75 \%$ or more of the population was Hispanic. The rate per 100,000 Hispanics was 63.1 ; for whites it was 75.4; and for Blacks it was 94.6 [Table 41].(305) Comparable rates per 100,000 women were 57.6 for Hispanics; 71.0 for whites; and 84.6 for Blacks.

Regional data for Puerto Ricans in New York indicate a slight excess stroke mortality rate compared to whites; this is particularly the case in the younger age-groups. (310)

Data based on 1980 census figures seem to indicate that the rate of stroke mortality decline for Mexican American men from 1970-1980 was half as steep as for white men in the same period(304), though the comparable rate of decline for Mexican American women was $1 / 3$ to $2 / 3$ steeper than for non-white women.(311)

## Hypertension

* 
* National data on hypertension morbidity in Hispanics are sparse. *
* Some regional data for Mexican Americans indicate that hyperten- *
* sion prevalence is greater than in whites, but lesser than in *
* Blacks, for men younger than 60 years of age. In older Mexican *
* American men, prevalence is increased, matching that in Black *
* men. In Mexican Americans, the rate of hypertension control for *
* women is similar to the national rate, but for men it lags far *
* behind.
* 
* 

There is ample and indisputable evidence of excess hypertension morbidity in Black Americans, but comparatively less information has
been available about this disease among Hispanics.
Hazuda, (304) Castro et al,(312) and Kumanyika and Savage(313) reviewed several studies that compared blood pressure levels and percentages of actual hypertensives in the population of Mexican American and non-Hispanic white men and women. These studies found diastolic blood pressures in men and systolic blood pressures in both men and women to be roughly comparable in both groups. $(314,315,316)$ The Laredo Project also assessed prevalence of elevated blood pressure ( $\mathrm{DBP}>95 \mathrm{~mm} \mathrm{Hg}$ ) among low SES Mexican Americans. Among men, the prevalence of elevated blood pressure in these Mexican Americans was intermediate between those of whites and Blacks in the Hypertension Detection and Follow-up Program (HDFP). Hypertension prevalence in Mexican American women, on the other hand, was lower than in either Blacks or whites in the HDFP. (314)

A comparison in these groups of prevalence of actual
hypertension (i.e. those with DBP $>95 \mathrm{~mm} \mathrm{Hg}$ and those with DBP $<95 \mathrm{~mm} \mathrm{Hg}$ but with either an history of hypertension or on antihypertension medication), the results showed that Mexican American men in Laredo, Texas had rates intermediate between the rates in whites and Blacks in the HDFP up to age 59 years. Older Mexican American men had rates of actual hypertension that equalled the rate of Black men in the HDFP. Mexican American women, however, had rates of actual hypertension that were slightly higher than in whites in the HDFP and, in the 60-69 year-old group, actually matched the rates in Blacks in the HDFP. These data also suggest that, like Blacks, Mexican American women in Laredo were more likely to have their hypertension controlled than were Mexican American men.(314)

Two other reports, one from a study in California, (317) and the other from the San Antonio Heart Study (318) presented data concerning gender and SES differences in hypertension prevalence in Mexican Americans. In the California study, which used elevated blood pressure as its criterion, there was a strong inverse relationship between SES and prevalence of elevated diastolic blood pressure in Mexican American men. This is consistent with trends observed in both Blacks and non-Hispanic whites. The overall prevalence of hypertension in Mexican American men was intermediate between that found in non-Hispanic whites and Blacks.

In the San Antonio Heart Study, which used actual hypertension as a criterion, there was no SES gradient in prevalence of actual hypertension in either Mexican American or non-Hispanic white men. For women, however, there was a strong inverse relationship between socioeconomic status and prevalence of actual hypertension in both ethnic groups. These data suggest that rates of hypertension appear to be comparable in Mexican American and non-Hispanic white men but lower in Mexican American women than in non-Hispanic white women. When adjustments for obesity are made, Mexican Americans tended to have lower rates of hypertension than non-Hispanic whites at comparable SES levels.

Hazuda et al(319) also reported data on the proportion of hypertensives previously diagnosed and under treatment, as well as the proportion under adequate control in the San Antonio Heart Study. The results showed that the proportion of previously diagnosed and
treated hypertensives was lower for Mexican Americans of both sexes than in non-Hispanic whites. Also, there was no SES gradient for Mexican American men, but among Mexican American women, the proportion of diagnosed and treated hypertensives actually decreased from low to high SES. No such SES gradient was observed for non-Hispanic white women, but there was a positive SES gradient for non-Hispanic white men (i.e. increased proportion of diagnosed and treated hypertensives with increased SES). In addition, the proportion of Mexican American hypertensives both under treatment and under control ( $\mathrm{DBP}<95 \mathrm{~mm} \mathrm{Hg}$ ) was lower for both sexes at the lower SES levels. In addition, the proportion of hypertensive Mexican American women under control was higher than for Mexican American men ( $87 \%$ vs $64 \%$ respectively). This difference was also seen in the control rates in the Laredo Heart Study (77\% for Mexican American women and only $37 \%$ for Mexican American men).(314)

Two other studies assessed hypertension knowledge and blood pressure care by physicians among Hispanics and found that $36 \%$ of Spanish-speaking residents of Arlington County, Virginia had adequate knowledge about hypertension risk, but most had less clear conceptions about high blood pressure and its etiology. (320) Ramirez, Hernick, and Weaver (321) also found in their survey of Mexican Americans in Houston that Mexican Americans had their blood pressures checked by physicians as often as non-Hispanic whites. However, fewer Mexican Americans were actually told their blood pressure readings, and most had misconceptions about high blood pressure.

Therefore, though hypertension is less prevalent in Mexican Americans than in Black Americans, the rates are still higher than those in non-Hispanic whites especially at the lower SES levels. More importantly, although the figures for Mexican American women are similar to national rates for hypertension control, Mexican American men lag far behind these rates.

## II: EXPLANATIONS FOR DIFFERENCES

[^1]* Limited regional data, for certain Hispanic subgroups only, are $\%$
* available on the impact of major biological risk factors (as *
* identified for white populations) on coronary disease. Caution *
* must be used in interpreting these data: not enough is yet known *
* about risk profiles in non-white populations.


Risk Factors



* Limited regional data indicate there is a higher prevalence (gen-* * erally) of obesity, noninsulin-dependent diabetes, hypertension, * * and high LDL-cholesterol levels in certain Hispanic subgroups $\%$ * than in whites. However, available data seem to indicate the risk* * of CHD in Hispanics to be lower than in whites, though such data * * is scant.
※

Two recent papers on coronary heart disease in Hispanics (304,312) provide an excellent review of the available evidence on cardiovascular risk factors in Mexican American and Puerto Rican populations. The majority of the studies reviewed reported data on Mexican Americans in California and Texas, and a few were based on Puerto Ricans in Puerto Rico and New York. The authors of both review papers note that mortality and morbidity from CHD would be expected to be higher in Hispanics than in whites given the strong association between low-income status and risk for CHD, and the over-representation of Hispanics in low-income, urban groups. Yet the available evidence suggests, at least for Mexican Americans and Puerto Ricans, that the rates of CHD mortality are lower than in whites. Both papers hypothesized that the observed trends may be due to sociocultural and risk factor differences between Hispanics and whites, some of which may confer some degree of CHD protection on Hispanics. Also, since the pattern of CHD mortality and morbidity is not uniform across gender/nationality/age-groups of Hispanics there may well be differences in risk factors between these groups.

The data bases that established the relationship between certain individual factors and subsequent CHD are primarily from white populations $(322,323)$, thus limiting our understanding of
(1) the relationship between known cardiovascular disease risk factors and the prevalence of CHD among Hispanics $(324,304)$;
(2) the relative predictive significance of each risk factor to CHD mortality and morbidity in Hispanics as compared to whites and other minorities;
(3) the possible existence of group-specific factors which may confer some protection against $\mathrm{CHD}(324)$ or increase the risk for same. (323)

Nevertheless, the available evidence on four major biologic risk factors for CHD is reviewed here, namely: triglycerides, lipoproteins and cholesterol in abnormal amounts; hypertension; obesity; and diabetes. Limited data have been obtained only on Mexican Americans and Puerto Ricans. Any conclusions that may be drawn for those two these groups should not be assumed to apply to the other Hispanic groups.

## (i) Lipids and Lipoproteins

Hazuda(304) and Castro et al(312) reviewed five studies on Mexican Americans and one study on Puerto Ricans that compared these Hispanics to non-Hispanic whites on levels of cholesterol and triglycerides. These studies typically show that Mexican Americans, especially low-income men, tend to have higher age-adjusted serum cholesterol levels $(314,318,316)$ and higher overall percentages of hypercholesterolemia (cholesterol>260mg/dl) in the population than non-Hispanic white men. (317) On the other hand, Mexican American women typically have cholesterol levels comparable to those of non-Hispanic white women. $(316,318)$

Studies that compared cholesterol levels between Hispanics and non-Hispanic whites as a function of social class found no evidence of the inverse relationship between SES and cholesterol level in Hispanics found in whites.(317) However, Friis et al(316) found that cholesterol levels in Mexican American men were comparable at the lower SES levels and increased dramatically in the high SES group. In both Hispanic and non-Hispanic white women, cholesterol level was found not to be related to SES.

In contrast, data on Puerto Rican men, ages 45-64 years, compared to white male cohorts in the Framingham study, found that the $P R$ men had diets that were lower in total calories, total cholesterol, saturated fats, and alcohol, and higher in complex carbohydrates.(325) These differences are similar to those obtained from rural vs urban men in Puerto Rico.(326)

Studies that assessed triglyceride levels found consistent and significantly higher triglyceride levels in Mexican Americans than in non-Hispanic whites. $(315,314,318,316)$ A positive relationship between SES and level of triglycerides was observed in Mexican American men but not in Mexican American women.

Friis et al(316) also examined the relationship between low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and social class in Mexican Americans in the San Antonio Heart Study. Their results paralleled the relationship between SES and total cholesterol. In Mexican American and white men, LDL-C was low at the lower SES levels and increased significantly at the upper SES levels. For women in both groups, LDL-C levels were comparable across SES levels. For HDL-C, the levels were the same at all SES levels in both Mexican American and white men, but increased significantly with SES and were similar in both Mexican American and white women. Therefore, the apparent lower CHD mortality rate in Hispanics does not appear to be due to differences in HDL-C.(327)

Two reports from the San Antonio Heart Study noted that, in Mexican Americans of both sexes, avoidance of fats and cholesterol in their diets increased significantly with SES. (328) However, Mexican Americans were less informed and less likely to attempt dietary modifications to reduce risk of heart disease than non-Hispanic whites. Health promotion information and behaviors did increase with SES in Mexican Americans.(319)
(ii) Hypertension
*W,

* Although overall prevalence of high blood pressure appears to be *
* lower in Hispanics than in Blacks and in whites, certain Hispanic*
* subgroups seem to have more hypertension: Puerto Rican and Cuban * * men and women.

The data previously reviewed showed that although the prevalence of high blood pressure appears to be lower in Hispanics than in Blacks and whites, several specific age/gender/nationality subgroups of Hispanics had excesses of hypertension, for example Puerto Rican and Cuban men and women. Also, reports from the Laredo and San Antonio Heart Studies (304) indicated that hypertension prevalence, whether assessed as proportion of the population with elevated blood pressure or as proportion with actual hypertension, was highest in low-income Mexican American men, especially after age 60. At this age, Mexican American men in Laredo, Texas had hypertension rates comparable to those found in the HDFP Black sample. This group was also least likely to have high blood pressure diagnosed and under control.

There was evidence of an SES gradient in hypertension risk in Hispanics similar to that found for Blacks and whites. This risk gradient appears to be mediated by obesity especially in Hispanic women, and limited knowledge about hypertension appears to increase the overall risk for hypertension and perhaps other CHD-related diseases among Hispanics.

## (iii) Diabetes Mellitus



* Noninsulin-dependent diabetes mellitus is a major health problem * * in Hispanics, especially in Mexican Americans and Puerto Ricans. * * However, the relationship between this risk factor and CHD has * * not been adequately studied in all of the major Hispanic groups. *


Noninsulin-dependent diabetes mellitus (NIDDM) is recognized as a major health problem for Mexican Americans, especially those from low socioeconomic backgrounds. $(318,329)$ The prevalence of NIDDM in low SES Mexican Americans ranged from $8.3 \%$ in both sexes in Starr County,

Texas (329) to $15.7 \%$ in men and $16.1 \%$ in women in Laredo, Texas. $(314,318)$ These rates are significantly higher than those of the general U.S. population.

Though there are very few studies that have investigated the role of diabetes in coronary heart disease in Puerto Ricans, the San Antonio Heart Study and Laredo studies, and the Diabetes Alert Study in Texas have collected extensive data on this problem in Mexican Americans. These studies identify low SES Mexican American women and men as running the highest risk of NIDDM, and that NIDDM prevalence declines in both sexes and in both Mexican Americans and non-Hispanic whites as SES increases.(318) Additional analyses suggest that the inverse relationship between SES and NIDDM may be mediated through obesity in Mexican American women, but not in Mexican American men. (330) Furthermore, reports from the National Diabetes Data Group (331) suggest that increased acculturation resulted in consistent reductions in NIDDM prevalence in both Mexican American men and women. This effect of acculturation on NIDDM, like SES, appeared to be mediated through obesity in Mexican American women but not in Mexican American men.

Additional analyses to determine whether the excess prevalence of diabetes in Mexican Americans could be due to obesity or to some other, perhaps genetic, factor yielded mixed results. Stern et al(332) found when comparing Mexican Americans and non-Hispanic whites (controlling for degree of obesity) that the prevalence of NIDDM was still significantly greater in Mexican Americans than in whites. Preliminary evidence has also been presented suggesting that the excess NIDDM prevalence in Mexican Americans that is not attributable to obesity may be related, instead, to degree of Native American admixture. Declines in NIDDM rates were observed as both SES increased and percentage of Native American admixture decreased in Mexican Americans. $(333,334)$

## (iv) Obesity



* Some data suggest that Mexican Americans have an excess preval- *
* ence of obesity compared to non-Hispanic whites; further, that *
* social mobility and increased acculturation seem to reduce this *
* risk more in Mexican American women than in Mexican American men.* *W*

Available evidence indicates that obesity is a major problem in Mexican Americans, especially among women and those of low socioeconomic status. $(314,315,318,335,336,337,338)$ Compared to the general U.S. population in which the prevalence of obesity obtained from NHANES I was $15.6 \%$ in men and $29.0 \%$ in women(339), Mexican Americans in the Laredo Study were reported as having an age-adjusted prevalence of obesity (i.e. $20 \%$ above ideal weight) of $25.8 \%$ in men and $44.8 \%$ in women. (314) Mueller et al(336) in their study of low-SES Mexican Americans in Starr County, Texas found obesity prevalence rates of $30 \%$ or higher in adults of both sexes.

Data from the San Antonio Heart Study also identified a significant gender-SES trend whereby body mass index (i.e. weight/height) decreased slightly with increased SES in Mexican American men but decreased dramatically with increased SES in Mexican American women. Comparisons within SES-matched strata still found excess adiposity among Mexican Americans of both sexes.(318)

Studies on dietary beliefs and attitudes noted that more Mexican Americans at each SES level were likely to express the belief that Americans are too concerned with losing weight, and Mexican Americans in both low and upper SES levels were less likely than non-Hispanic whites at comparable levels to avoid sugar or to diet. (332) These differences were especially significant for women in the low SES groups.

A recent report by Hazuda et al(330) found a significant decrease in body mass index in Mexican Americans of both sexes as level of acculturation increased.

Finally, there has been increased interest recently in the hypothesis that the distribution or patterning of body fat may be an important determinant of metabolic disorders such as diabetes mellitus, which may be related to heart disease. Limited evidence suggests that Mexican Americans have relatively more central distribution of body fat than non-Hispanic whites (338,340), and that body fat in Mexican American men increases with increasing SES though it decreases with increased SES in Mexican American women. (338) Other evidence suggests there is more upper body fat than lower body fat in Mexican American diabetics as compared with non-diabetics. $(336,337)$

## (v) Cigarette Smoking

* 
* What data there are indicate that, though more Hispanics smoke $\quad$. * cigarettes, fewer are heavy smokers. There are some indications, * * however, that there is heavier smoking among Hispanic youth com- * * pared to white and Black youth. Little is known about the impact * * of smoking by Hispanics on CHD risk.


Hazuda(304) reviewed six studies that compared smoking behavior in Mexican Americans and non-Hispanic whites, and Castro et al(312) reviewed two additional studies on smoking in Mexican Americans and one study on Puerto Ricans. These studies suggest that
(i) the overall rate of current smoking appears to be approximately the same or slightly higher in Mexican Americans than in whites, with the proportion of Mexican American men smoking the same or more than white men and the proportion of current smokers lower among Mexican American women than among white women $(316,335,341)$;
(ii) these trends hold, regardless of socioeconomic status; and
(iii) Mexican American smokers smoke significantly fewer
cigarettes per day than non-Hispanic whites. $(314,335,341)$
Recent reports on smoking among Black, white, and Mexican American youth, however, show a marked increase in smoking among Mexican

American youth, and more of these youth smoke than their Black and white peers (i.e. $28.9 \%$ vs $15.2 \%$ and $19.1 \%$, respectively). ( 342,343 )

One San Antonio Heart Study report looked at how well-informed subjects were about the role of smoking as a risk factor for heart disease and whether they had modified their smoking behavior to reduce their heart disease risk.(319) The results showed that Mexican Americans of both sexes and low SES Mexican Americans were significantly less well-informed than non-Hispanic whites or than their higher SES peers, respectively. Mexican American men were better informed about the risks of cigarette smoking than Mexican American women.

In Puerto Ricans, Sorlie et al(344) found that middle-aged men smoked less than comparable cohorts in the Framingham study. Only very limited information is available on smoking in other age, gender, and SES groups for Puerto Ricans, Cubans, and other Hispanics.

## B: Socioeconomic and Sociocultural Factors



* Among Mexican Americans, those with lowest SES and level of *
* acculturation have significantly worse cardiovascular risk factor*
* profiles than those in higher SES groups. Mexican American women $\stackrel{\downarrow}{*}$
* appear to gain more from increased acculturation and social mob- *
* ility than do men. Limited comparable data on other Hispanic *
* groups precludes any generalization to these other groups. *
* 


## Socioeconomic Factors

The major reported socioeconomic factors are socioeconomic status (SES), social mobility, and level of acculturation. Hazuda(304) and Castro et al(312) report improvements in the social status of Mexican Americans as shown by increases in the numbers graduating from high school and from college, by increases in occupational status as more Mexican American men are employed in nonagricultural jobs, and more Mexican American women entered the labor force. These improvements, although noteworthy, are not significant enough to produce any meaningful upward shifts in social status of Mexican Americans overall, relative to non-Hispanic whites. The same could probably be said of Puerto Ricans(345), Cubans, and other Hispanics.(303)

Nevertheless, Hispanics are almost certainly becoming increasingly more acculturated as reflected in the percentage of first-, second-, and third-generation Mexican Americans and the rate of outmarriage among Mexican American women. In the five southwestern states in 1970, which contain about $90 \%$ of all Mexican Americans, $12 \%$ were first-generation Americans, $30 \%$ were second-generation, and $58 \%$ were third-generation Americans. $(345,346)$ The overall incidence of outmarriage is low among Hispanics and lowest among Mexican Americans. However, the trends show that younger, more educated Mexican American women have the highest outmarriage rates. These trends are likely to be similar for the
other Hispanic groups. Thus, Hazuda(304) notes, the Mexican American population is becoming somewhat more heterogeneous socioeconomically and culturally, but the majority of Mexican Americans remain in the lower SES strata, and experience low-to-intermediate levels of acculturation in American society.

## Sociocultural Factors

The contribution of sociocultural factors such as health beliefs, attitudes, and behaviors (i.e. illness behaviors) to increased CHD risk or to greater resistance to CHD is of interest. Two recent papers by Schreiber and Homiak(345) and Harwood(301) on Mexican Americans and on mainland Puerto Ricans discuss several important sociocultural features of the health beliefs, pattern of symptom expression and meaning of illness, and illness behaviors that distinguish these groups from whites and other ethnic minorities. In both papers the authors acknowledge the importance of "folk classifications" and interpretations of illnesses (e.g. "Empacho", "Mal Ojo", "Susto") and of culture-specific disorders (e.g. "Ataque") that are based on a "spiritist or animistic" etiology and which are treated by folk remedies and procedures. These beliefs and practices are expressions of the culture, and reflect a more naturalistic and humanistic cultural ideology. This is in stark contrast to the more mechanistic disease perspective which is the foundation for the high quality of health care prevalent in the United States. One of the natural consequences of this naturalistic view of health and illness is more widespread use of folk remedies and of folk healers, especially among the less affluent, more rural, less acculturated, and older Hispanics. Therefore, interventions targeted for these groups need to be responsive to these sociocultural beliefs and practices, and the differences between traditional and modern health care.

The need for attention to cultural beliefs and practices should not be interpreted as suggesting that these beliefs and practices are widespread thoughout all Hispanic groups and present only problems. Rather, they exist to some degree in various Hispanic subgroups, might contribute to increased resistance to modern health care practices, or, on the other hand, might confer some protection against CHD (e.g. through diets low in saturated fats and sodium). In addition, social class, level of acculturation, and nationality may also influence the degree to which Hispanics adhere to folk beliefs and practices.

## C: Behavioral and Sociocultural Factors

## Introduction

Some of the factors that may affect risk for CVD are themselves behaviors, such as cigarette smoking and lack of physical exercise. Other known and/or suspected risk factors are, themselves, affected by behaviors in positive or negative ways. In addition, among Mexican

Americans (for example), these risks are modified by social class, gender, and acculturation such that the most negative behavioral risk profile is found in the least affluent men in the population. This is true whether we look at the behavior in question (i.e. smoking, exercise) or at knowledge of the relative risks or benefits of these behaviors. The most beneficial behavioral risk profile in Mexican Americans appears to be in the more affluent, upwardly mobile and acculturated women.

The relative paucity of data on Puerto Ricans, Cubans, and other Hispanics preclude any conclusions about whether these health behaviors might account for differences in cardiovascular disease risk between these groups and whites.

## Exercise

Hazuda(304) reviewed three studies that compared level of physical exercise in Mexican Americans and non-Hispanic whites, and noted that all three studies concluded that there was a lower level of physical exercise during time not spent at work in Mexican Americans than in non-Hispanic whites. $(316,319,336)$ Hazuda et al(319) also reported that non-Hispanic whites were better informed about exercise as a potential reducer of CHD risk and reported more preventive behaviors than Mexican Americans. However, both knowledge and actual exercise frequency increased with increasing SES among both Mexican American men and women. These data suggest that Hispanics lag significantly behind non-Hispanic whites both in knowledge about and in regular practice of exercise as a CHD risk reduction activity.

## D: Access to and Utilization of the Health Care System

The less affluent Hispanics run significantly greater CVD risks than more affluent Hispanics. This picture of greater relative vulnerability in the poor holds true regardless of ethnic group or gender, and probably results not only from greater prevalence of biological and sociocultural risk factors but also from less frequent or less effective access to good quality medical care. (345) Although most of the medical evidence points to individual, social class, and ethnic group factors as primary causes of the poor health of these groups, there is also evidence that health system factors and social policy decisions also contribute to the observed health status differences. Recent papers have suggested that reductions in health services have negative effects on the quality and effectiveness of the medical care. (347,348,349) Another general review of the literature(350) on access to health care by the poor reaffirms the existence of a significant disparity in a wide range of medical services between the poor and the non-poor.

# CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN ASIAN/PACIFIC ISLANDER AMERICANS 

## I INTRODUCTION

The classification of Asian/Pacific Islander is a demographic category that subsumes a variety of ethnic, cultural, and national groups. Included are such ethnically and culturally diverse groups as Japanese, Chinese, Koreans, Filipinos, East Indians, Polynesians, and southeast Asian refugees (e.g. Cambodians, and Vietnamese). Though these groups may be roughly described as sharing some Eastern cultural and ethnic characteristics, they may well be as different from each other as they are from whites, Blacks, Hispanics, and Native Americans. (400) Also, within each group, members vary in terms of immigration status (i.e. immigrant vs citizen), nationality (i.e. native-born vs foreign-born), and level of acculturation (i.e. among the native-born, varied generations since migrating to the U.S.). In addition to these problems, this review was severely hampered by the limited availability of reliable information on even a single, reasonably homogeneous Asian population, much less any on the diverse groups that make up this classification.

In a recent report, Yu et al(400) note that Asians are the largest growing segment of the U.S. population. According to the 1980 census, Asians have grown faster than all other ethnic groups (i.e. 120\% growth for Asians vs $6.4 \%$ for whites, $17.4 \%$ for Blacks, and $60.8 \%$ for Hispanics). This extraordinary growth pattern can be attributed to immigration, high birth rates, and to the redefinition of this census category to include other groups (e.g. southeast Asian refugees). The largest Asian group are the Chinese ( $23.4 \%$ of the Asian population), the Filipinos (22.6\%), Japanese (20.7\%), East Indians (11.2\%), Koreans (10.3\%) and Vietnamese (7.1\%). (400)

As a group, Asians are disproportionately concentrated in the West, they are primarily an urban people ( $92 \%-97 \%$ of Asians are urban vs $71 \%$ of whites), and they are one of the most highly educated groups in the U.S. (i.e. roughly $30 \%$ of Asians have a college degree vs $17.5 \%$ of whites). However, this higher educational attainment has not been translated into comparably higher occupational status since Asians hold professional occupations only about as frequently as the lesser-educated white population. Foreign immigration affects the concentration at both ends of the occupational distribution because foreign-born Asians are both more likely to be service workers as well as more likely to be professionals than native-born Asians. This suggests that Asian immigration includes two groups from distinctly differing socioeconomic strata. Among Asians, the occupational status distribution also varies across groups, with Filipinos and East Indians over-represented among professionals and the other groups distributed more evenly along the occupational continuum.

The income profile of Asians is also quite complex. Although the 1980 Census data show that, regardless of nativity, Asians have a
higher median family income than whites, other adjustments in these data which take household size into account, for example, suggest that, in addition to the Vietnamese and other refugee groups, the Chinese and Korean Americans also have a prevalence of poverty that is above the national average [Tables 50-52]. $(400,401)$

Most of the relevant epidemiologic and other research information places all Asians in one group (NCHS data), with occasional distinctions made between Japanese, Chinese, and Hawaiians (e.g. The Honolulu Heart Study), and to a lesser degree Koreans and Filipinos (e.g. The Los Angeles County Cardiovascular Diseases data, and unpublished data from NCHS). More recently, increased attention is being given to the health and other needs of southeast Asian immigrants (e.g. The Indochinese Health and Adaptation Study, San Diego, California), but the available information on this population is mainly descriptive of their socioeconomic and general health status and needs with none on to coronary heart disease and risk status.

Because of the diversity of these groups, the significant percentage of recent immigrants among them, and the tendency of many of these groups to cluster in defined neighborhoods where their primary language and culture flourish and where outgroup suspiciousness is common, it has been difficult to obtain accurate Census figures or to reliably assess health needs, health habits, attitudes toward health care, or pattern of health care utilization. Differing reports have been made suggesting that regular reliance on folk medicines is used as a substitute for or a complement to Western medical services. This pattern of health care is believed to be especially prevalent among the elderly, more recent immigrants and the less acculturated members of these groups.(402) Such claims, however, should be treated with caution because many are based on flawed studies or rely too heavily on anecdotes and impressions.

Because of the limited and uneven availability of data on the health of Asians, data on cardiovascular disease morbidity, mortality, and risk status of Asians as a group will be presented and, where available, also discussed for specific major Asian/Pacific Islander subgroups.

## Coronary Heart Disease

## (i) Mortality

## 

* Heart disease is the leading cause of death for all American $\quad *$ * Asian groups. Some regional data seem to indicate that Asians are* * at lower risk of mortality from vascular disease than whites $\quad \underset{~}{~}$ * and than other minorities, with the possible exception of stroke. ${ }^{*}$ * Asian women appear to be at lower risk than men in all ethnic * * subgroups. Koreans, Filipinos, and Chinese appear to be at lower * * risk of cardiovascular disease than Japanese men and women. *

higher median family income than whites, other adjustments in these data which take household size into account, for example, suggest that, in addition to the Vietnamese and other refugee groups, the Chinese and Korean Americans also have a prevalence of poverty that is above the national average [Tables 50-52]. $(400,401)$

Most of the relevant epidemiologic and other research information places all Asians in one group (NCHS data), with occasional distinctions made between Japanese, Chinese, and Hawaiians (e.g. The Honolulu Heart Study), and to a lesser degree Koreans and Filipinos (e.g. The Los Angeles County Cardiovascular Diseases data, and unpublished data from NCHS). More recently, increased attention is being given to the health and other needs of southeast Asian immigrants (e.g. The Indochinese Health and Adaptation Study, San Diego, California), but the available information on this population is mainly descriptive of their socioeconomic and general health status and needs with none on to coronary heart disease and risk status.

Because of the diversity of these groups, the significant percentage of recent immigrants among them, and the tendency of many of these groups to cluster in defined neighborhoods where their primary language and culture flourish and where outgroup suspiciousness is common, it has been difficult to obtain accurate Census figures or to reliably assess health needs, health habits, attitudes toward health care, or pattern of health care utilization. Differing reports have been made suggesting that regular reliance on folk medicines is used as a substitute for or a complement to Western medical services. This pattern of health care is believed to be especially prevalent among the elderly, more recent immigrants and the less acculturated members of these groups. (402) Such claims, however, should be treated with caution because many are based on flawed studies or rely too heavily on anecdotes and impressions. Because of the limited and uneven availability of data on the health of Asians, data on cardiovascular disease morbidity, mortality, and risk status of Asians as a group will be presented and, where available, also discussed for specific major Asian/Pacific Islander subgroups.

## Coronary Heart Disease

(i) Mortality
N.

* Heart disease is the leading cause of death for all American * Asian groups. Some regional data seem to indicate that Asians are* $*$ at lower risk of mortality from vascular disease than whites $\quad *$ * and than other minorities, with the possible exception of stroke. $\%$ $\pm$ Asian women appear to be at lower risk than men in all ethnic * * subgroups. Koreans, Filipinos, and Chinese appear to be at lower * * risk of cardiovascular disease than Japanese men and women. $\quad \%$


National vital statistics' data identify Asians as a single group, and are quite limited, but suggest that the mortality rate relative to whites is approximately the same for heart disease, ischemic heart disease, cerebrovascular disease, and for the related hypertensive disease and diabetes. No significant gender differences were observed [Tables 53-57]. A pattern of relatively lower risk for CHD comes from reports from the Honolulu Heart Study $(403,404,405,406)$, and the Los Angeles County mortality data. $(407,408)$

Yu et al(400) compiled unpublished national mortality data from NCHS for 1980 that compared mortality from the 10 leading causes of death in whites, Chinese, Japanese, and Filipinos [Tables 58-59]. These data show that heart disease is the leading cause of death for all Asian groups, and that stroke is the third leading cause of death. Atherosclerosis is the tenth leading cause of death in the Asian groups, and is ninth among whites. Also, among the three Asian groups compared, these vascular diseases account for comparable proportional mortality (i.e. 31.8 for Chinese, 30.4 for Japanese, and 33.5 for Filipinos). Age-adjusted ratios for cardiovascular mortality rates in Chinese and Japanese are consistently higher than in Filipinos, and the rates in Chinese are typically higher than in Japanese. Japanese and Filipinos are equally at risk for heart disease.

Yu et al also computed age-adjusted, sex-mortality ratios (i.e. age-specific death rates for men divided by age-specific death rates for women) for whites, Chinese, Japanese, and Filipinos and found that men were at greater mortality risk overall than women in all ethnic groups. They also observed, however, that the groups did not differ markedly in these ratios (i.e. whites $=1.82$, Chinese $=1.75$, Japanese $=1.65$, and Filipinos $=1.96$ ) [Table 60].(400) Across groups, foreign-born Asians appear to have a disproportionate excess mortality compared to those born in the United States. These differences may be due in part to the distinctive health status and health habits of different cohorts who migrated to the U.S. at different times (See Table 19, Yu et al, 1984; reference 400).

In the Heart Association Report on Cardiovascular Disease Mortality in Los Angeles County(408), heart and cerebrovascular diseases were among the five leading causes of death for all groups compared, including Asians. However, subtle but important differences between the groups were observed in terms of the relative ranking of cardiovascular diseases. Among Japanese and Chinese, heart diseases were the primary cause of death (32\% of all deaths in both groups). However, among Koreans, malignant neoplasm was the leading cause of death ( $25 \%$ of total deaths), and heart disease ( $16 \%$ of all deaths) was second. For both Japanese and Chinese, CVD ranked third among the leading causes of death ( $15 \%$ and $11 \%$ of all deaths respectively) [Table 61].(400)

When we look at age-adjusted mortality/100,000 population for 1979-1981 for each of the ethnic/gender groups, we find that the mortality rates for both men and women in all Asian groups were significantly lower than in the other ethnic/gender groups for all causes and for most cardiovascular diseases. Among the Asians, there
was a consistent trend toward lower mortality rates in Koreans and Chinese, and slightly higher rates in Japanese. This group differential also held true for men and women.

In an earlier report on cardiovascular disease mortality in L.A. County(407), data were reported for Filipinos. These data show that the annual mortality rate $/ 100,000$ in 1980 was lower for Asians than all other races for major cardiovascular diseases. Within the Asian group, the Koreans and Filipinos had the lowest rates, the Chinese had higher rates, and the Japanese had the highest rates. For hypertension, the mortality rates for the Chinese were slightly higher than the other Asian subgroups. Filipinos had the lowest age-and sex-adjusted mortality rates of all the subgroups. Yu et al(400) suggested that the relatively higher socioeconomic status of this population due to the disproportionate migration of educated Filipino professionals might account for the relative "resilience" of the population as compared to other groups with a broader socioeconomic status distribution. Mortality rates for Japanese for all types of cardiovascular disease, though lower than for whites, Blacks, and Hispanics, were the highest of all Asian groups [Table 62]. (407)

Interesting age and gender differences in mortality rates are also observed between Filipinos and Koreans, with Filipino men and women between ages 45-54 having higher rates than their Korean cohorts. The total Filipino male mortality rate for major CHD is higher than that for Koreans, though the reverse is true for women.

In the case of heart disease, once again the mortality rates for the Japanese exceed rates for all of the other groups for both men and women, and at most age levels. There were no significant differences in heart disease mortality between Chinese and Filipino men, and Korean men had the lowest mortality rate. For women, the mortality rate in the Chinese was lower than in the Japanese but significantly higher than the rate for Korean and Filipino women [Table 63]. (407) Similar trends across age, gender, and nationality groups were also observed for ischemic heart disease [Table 64]. (407)

Data from several sources on CHD mortality for Chinese men, ages 35-74 years, in Hawaii $(409,410)$ showed CHD rates lower than for whites but higher than for Japanese. On the other hand, Chinese women were at higher risk than women of all other ethnicities except Hawaiians. $(410,411)$

Trends in CHD mortality in Japanese men in Hawaii showed significant increases between 1940 and 1970 , but declined somewhat between 1970 and 1978. Since 1970, there have been declines in CHD mortality rates in women of all ethnic groups except Hawaiian and Filipino women who showed declines since 1960.

Gerber and Madhavan(411) compared proportional mortality due to CHD among Chinese in Hawaii, native and foreign-born Chinese in New York City, and whites in New York City between 1968 and 1972. CHD deaths were proportionately higher in Chinese in Hawaii vs those in New York City in every age-group with the size of the difference narrowing with increasing age and disappearing in the 75+ age-group. Among the Chinese in New York City, CHD deaths were proportionately greater in U.S.-born vs foreign-born Chinese at all ages 25 and over.

A lower proportion of deaths was due to CHD mortality among Chinese in both Hawaii and New York City vs whites in New York City, except in the 25-44 year age-group where proportionate mortality was higher in Hawaiian Chinese. Death from CHD occurred later in Chinese populations than in New York City whites and later in foreign-born New York City Chinese than in the other two Chinese subgroups. These findings are consistent with an increasing and earlier CHD risk with increasing U.S. exposure (i.e. acculturation).

It has been suggested that the overall higher socioeconomic status of Asians as a group may partially account for their more favorable cardiovascular status. (400) However, within-group differences in SES and nativity need to be explored to determine whether there are particular subgroups of Asians who are at risk for excess cardiovascular mortality (e.g. recent Chinese immigrants with low SES). The Japanese, who generally are more acculturated to U.S. lifestyles, diets, etc., appear to be at higher CHD risk. $(406,412,413)$
(ii) Morbidity


* Data on nonfatal events are too sparse for any conclusions to be * $\div$ made about incidence, prevalence, or trends.
以


## Stroke

(i) Mortality


* National data suggest stroke mortality rates in Asian Americans $\%$
* are similar to whites; that stroke is the third leading cause of *
* death. Recent age-adjusted data for Asian subgroups indicate *
* that Japanese men are unique among most ethnic/gender groups, $\quad \div$
* including whites, in having the highest stroke mortality rates. * *

National data, which identify Asian Americans as a single group, are quite limited, but suggest that the mortality rate for cerebrovascular disease is similar to that in whites. (425) More recent (unpublished) national data, compiled by Yu et al(400) and which identify Chinese, Japanese, and Filipino Americans separately, indicate that stroke is the third leading cause of death in these groups; and that stroke accounts for a slightly higher proportion of all-cause mortality for Japanese (11.2) and Filipinos (10.1) than for Chinese (8.6). The proportion for whites is the same as for Chinese (8.6). Age-adjusted mortality ratios (i.e. minority rates compared to white rates) showed that Japanese and Chinese Americans are equally at risk for cerebrovascular death. (400)

The Heart Association Report on Cardiovascular Disease Mortality in Los Angeles County (408) indicates that cerebrovascular disease was
third among the leading causes of death in Asians in general. Within different ethnic Asian subgroups, there were some differences, however. Japanese had the highest rates expressed as a percentage of all-cause mortality ( $15 \%$ ), and Chinese and Korean Americans had lower, approximately equal, rates ( $11 \%$ and $10 \%$, respectively). Age-adjusted mortality rates for $1979-1981$ reveal that most Asian age/gender groups have lower rates for all-cause and for most vascular diseases than the other American ethnic/gender groups. A notable exception, however, is found in Japanese men, who have the highest mortality rates of most groups, Asian and non-Asian, for cerebrovascular disease ( 86.7 deaths $/ 100,000$ population). This is higher than in whites (75.4), higher than in Hispanics (63.1), and higher than the United States overall stroke mortality rate of 63.1/100,000 population.

## (ii) Morbidity

Some limited data suggest that important differences in the incidence and prevalence of stroke exist among Japanese living in Japan, Hawaii, and California.(414) The prevalence of stroke was significantly higher among Japanese living in Japan (age-adjusted rate $=35.4$ ) as compared to Japanese in Hawaii (10.7) and in California (10.4) [Table 65]. (415) The same is true when the annual incidence rates are compared for Japan and Hawaii (7.4 vs 2.7) [Table 66]. (416) However, there are no significant differences between these two groups of Japanese with respect to predominant type of stroke. In Japanese in both Japan and Hawaii, thromboembolic stroke predominates over intracranial hemorrhage [Tables 67-68].(416)

## II: EXPLANATIONS FOR DIFFERENCES

## 

* With few gender/subgroup exceptions, the limited data that exist * * suggest that mortality risk for cardio- and cerebrovascular dis- * * ease is lower in Asians than in whites, or similar. Data on risk * * factor profiles, SES levels, and acculturation are too limited $\Rightarrow$ * to date for generalizations to be made. $\quad \Rightarrow$
* 

The limited data available suggest that mortality risk for cardiovascular disease is lower in Asian groups than in the white population. Assuming that these data are valid and apply to all Asian groups, then we are faced with the question of what accounts for this difference. Comparatively lower CHD risk factor levels might explain this difference. Similarly, the differential risk for stroke vs heart disease may also be related to different risk factor profiles. Yu et al(400) also suggest that there are sociocultural attributes inherent in Asian cultures and lifestyles, and in the above-average socioeconomic status Asians have achieved that might
confer a significant degree of protection against CHD on these groups as compared to whites and the other ethnic groups. Unfortunately, the available research on cardiovascular disease risk factors in Asian populations is based primarily on studies of the Japanese in Japan, Hawaii, and California (The Ni-Hon-San Study), and on Japanese and Chinese in the Honolulu Heart Study and in the State of California Heart Survey. A few of these studies also addressed risk factors in Filipinos and Hawaiians. Therefore, generalizations of these findings to other Asian groups are unjustified and probably unwise.

## A: Biologic and/or Physiologic Variables in Japanese Americans

## Hypertension



* Prevalence of hypertension in Japanese Americans appears to be $\%$
* lower overall than in whites, and Japanese men appear to be less * * likely than white men to show evidence of ECG left ventricular * * hypertrophy. This pattern of low prevalence of hypertension, how- $\%$ $\%$ ever, is influenced by age, gender, sociogeographic area, and $\%$ * according to which generation is selected for use as an index of * * acculturation to the United States.

Two papers from the Honolulu Heart Study by Yano et al(417) and by Gordon et al(418) show that among these Japanese men, ages 45-64 years, the prevalence of hypertension (i.e. BP> or $=160 / 95 \mathrm{~mm} \mathrm{Hg}$ ) was relatively low (16.7\%), compared to whites. Gordon et al(418), comparing Japanese men in Honolulu with the Framingham men, noted a significantly higher prevalence of ECG-LVH in the Framingham sample than among the Japanese men ( $2.3 \%$ vs $0.7 \%$ ).

Comparisons of systolic blood pressure levels of Japanese men living in Japan, Honolulu, and California reported by Winkelstein et al(404) resulted in an overall impression that blood pressure levels of home-island Japanese men were intermediate between those of northern Californian Japanese men, who had the highest levels, and those of Hawaiian Japanese men. These data also identified a lack of concurrence between the distribution of blood pressures and the prevalence of stroke and heart disease in Japanese in the three areas. The Japanese men in Japan with moderate levels of blood pressures had high stroke rates, but low rates of heart disease. The Japanese in northern California, on the other hand, had the highest mean blood pressures and the highest rates of heart disease, but the lowest rate of stroke. The Japanese in Hawaii had the lowest blood pressure levels, but intermediate rates of both stroke and heart disease. In all three areas, Issei Japanese men (born in Japan but who had migrated to the U.S.), under age 55 years, had similar blood pressures; however, Californian Issei men over the age of 55 years had higher readings than their counterparts in the other two areas. Blood pressures levels for Issei cohorts over age 55 in Japan were
somewhat higher than those for Issei men of similar age in Hawaii. The differences in blood pressure levels among these cohorts were primarily explained by weight, although weight-blood pressure relationships were unstable in the men in Japan.

The California Hypertension Survey, (419) which reported both mean blood pressure levels and estimates of the prevalence of hypertension ( $B P>140 / 90 \mathrm{~mm} \mathrm{Hg}$ ), is a multistage probability sample of Asians in California. Among Californian Japanese men, ages 18-49 years, the prevalence of hypertension was slightly higher than in white men ( $19.2 \%$ vs $15.0 \%$ respectively). For men over age 50 years, the prevalence of elevated blood pressure among Japanese men by this criterion was lower than in white men ( $29.1 \%$ vs $38.5 \%$ ). Hypertension prevalence for Japanese women was much lower than for white women at all ages ( $0.4 \%$ vs $4.8 \%$ for those aged $18-49$ years, and $13.9 \%$ vs $36.4 \%$ for those more than 50 years old).

In 1979, levels of hypertension awareness among Japanese hypertensive men in California, ages 50 or more years, were higher than white men, but somewhat lower in Japanese women compared to white women. Younger Japanese men, ages 18-49 years, however, were less knowledgeable than their white male counterparts. (420) The proportion of Japanese male and female hypertensives under treatment as well as having their blood pressures under control was consistently smaller than in their white gender cohorts.

## Cholesterol

为
$\star$ The evidence on cholesterol suggests that, overall, Japanese men $\#$

* typically have lower total cholesterol levels and prevalence of *
* hypercholesterolemia than whites and, further, that Japanese in *
* Japan show consistently lower total cholesterol levels than their*
* Japanese cohorts in Hawaii and California.


For the younger segment of the Honolulu cohort, ages 45-54 years, Yano et al(417) report a mean level of baseline serum cholesterol of $219.4 \mathrm{mg} / \mathrm{dl}$ and report a $13.4 \%$ prevalence of hypercholesterolemia (cholesterol $>260 \mathrm{mg}$ ). Comparison of total cholesterol levels in Japanese men, ages 45-64 years, in the Honolulu cohort with those of Framingham men were reported by Gordon et al.(418) Methodologies for cholesterol determinations were sufficiently similar in the Honolulu and Framingham studies to allow direct although not exact comparisons. The results showed that serum cholesterol levels were approximately 15\% lower in Horiolulu than in Framingham men (a mean of $218.55 \mathrm{mg} / \mathrm{dl}$ vs $233.96 \mathrm{mg} / \mathrm{d} 1$, respectively).

A later report by Nichaman et al(414) which compared baseline serum cholesterol levels of Japanese men in Hawaii, Japan, and California found that, at all ages and for each biochemical variable compared, Japanese men in Japan had consistently lower values than those in Hawaii and in California [Table 69]. $(414,421)$

The prevalence of cigarette smoking in the Honolulu cohort of Japanese men, ages 45-54 years (417), was 46.4\%. The percentage of men smoking more than a pack of cigarettes per day was $19.2 \%$. In the comparison of 45-64 year-old Framingham and Honolulu men reported by Gordon et al(418), $44.1 \%$ of Honolulu men vs $57 \%$ of Framingham men were smokers. Robertson et al(422) compared baseline smoking status for Japanese men, ages 45-68 years, in Japan vs Hawaii and found a higher percentage in Japan of smokers ( $75.6 \%$ vs $44.2 \%$ ); however, they found more Japanese men who smoked 21 or more cigarettes a day in Hawaii than in Japan ( $16.6 \%$ vs $11.6 \%$ ). Taken together, these data indicate that Japanese men in Honolulu smoke more than Japanese men in Japan but less than white men in Framingham.

More recent estimates of cigarette use among Japanese Americans in California are available from the 1979 survey results. Overall, 50.6\% of the Japanese American men were classified as "ever smoked". (419) Fewer Japanese men and women described themselves as current regular smokers and as current or former smokers. Japanese smokers typically also smoke fewer cigarettes than their white male and female cohorts, but fewer Japanese than white smokers wanted to quit.

## B: Overall Impact of Risk Factors on CHD in Japanese Americans

为
$\star$ Though the standard risk factors for CHD are significantly less * $\star$ prevalent among Japanese on the mainland, in Japan, and in Hawaii* $\star$ than in white American men, generally similar associations are $\%$ $\star$ found between the major risk factors and fatal and nonfatal CHD, * * nonfatal MI, \& acute coronary insufficiency. High blood pressure,* $\star$ cigarette smoking, and high cholesterol levels are important $\quad \underset{*}{*}$

Reed et al $(405,426)$ presented total and fatal myocardial infarction (MI) incidence data for $50-59,60-63$, and $64-67$ year-old men in men of Japanese ancestry in Hawaii. cohort. Incidence rates for total MI increased overall between 1967-70 and 1975-78 for men, ages 60-67 years, and appear to have remained constant in the $56-59$ year-old men. Fatal MI rates showed a slight increase in the 60-67 year-old men with evidence of tapering off after 1971-74. Fatal MI rates were constant in the 56-59 year-old men [Table 70].(405) Analyses by birth cohort indicate upward slopes for total and fatal MI incidence during this time period in all cohorts of men born between 1900 and 1919.

Gordon et al(418) reported that, although the standard risk factor associations were observed among men in the Honolulu cohort, two-year CHD incidence (defined by ECG) was twice as large in the Framingham study, and CHD mortality was four times larger than in the

Honolulu study. Even after adjustments for differences in levels of blood pressure, smoking, and cholesterol, CHD mortality was larger by a factor of 2.1 in the Framingham study.

In a later analysis of six year follow-up data, Gordon et al(423) attempted to identify factors other than blood pressure, smoking, and cholesterol that could explain the differences between Framingham and Honolulu CHD rates. Alcohol intake was found to be an inverse predictor of MI and CHD death, although all-cause mortality rates increased as alcohol consumption increased. Higher starch intake in the Honolulu cohort was also noted as a possible protective factor for CHD. Elsewhere, the possible differential factor of later age of acquisiton of risk factors among Japanese migrants vs U.S.-born men has been noted as a possible explanation for CHD rates less than those predicted with Framingham logistic functions. (422)

Robertson et al(422) compared risk factor-CHD associations in Japanese men in Honolulu and Japan, and found that gradients for blood pressure and cholesterol on CHD incidence were similar in both cohorts of men. However, smoking was the most significant risk factor in Honolulu, whereas, in Japan, it was not related to CHD incidence. This suggests the possibility that cigarette smoking may be a less important CHD risk factor in men with low serum cholesterol levels. An influence of relative weight on CHD was also observed in Japanese men in Honolulu but not among the men in Japan, perhaps because of the relatively small number of obese men in the Japan cohort.

Multivariate analyses conducted by Yano et al(417) indicated that, in addition to blood pressure, cigarette smoking was next in line as a strong predictor of CHD in all categories except angina. Alcohol consumption was a strong, independently associated, protective factor for both fatal CHD and for nonfatal MI. Serum cholesterol was strongly associated with total CHD and nonfatal MI and significantly, but less strongly, related to fatal CHD. Relative weight was not independently associated with any of the CHD manifestations in the multivariate analysis.

## C: Biologic and/or Physiologic Variables in Chinese Americans

中
$\star$ From the limited data available on CVD risk factors in Chinese $\quad \%$ * Americans, a cautious conclusion would seem to be that they tend * * to have lower levels of the major CHD risk factors, identified in* * whites. However, the data are not entirely consistent: Chinese $*$ * men over 50 years of age may have a greater prevalence of smokers* * (who smoke fewer cigarettes per day, however), and of elevated $\%$ * blood pressure than white men.
$\pm$
※
Significantly less information is available on CHD risk factors for Chinese Americans than for Japanese. Nevertheless, there are limited data from the Hawaii Heart Study sufficient for some preliminary
impressions about the relative CHD risk status of Chinese Americans relative to whites and to Japanese Americans to be made.

A component of the Hawaii Cardiovascular Study compared cardiovascular risk factors in 30 Chinese and 68 Japanese male MI survivors with those of CHD-free controls in 1966 and 1967.(424)

The Chinese men were more obese than the Japanese as defined by skinfold tests, but were not more overweight by the Quetelet index. Serum cholesterol levels of the Chinese population controls were approximately $20 \mathrm{mg} / \mathrm{dl}$ higher than those of the Japanese controls (242.7 vs 220.7). Blood pressure levels and LVH patterns were not consistently or significantly different between the two racial groups. A higher proportion of Japanese were smokers ( 75 vs $70 \%$ ) as compared to Chinese in the immediate pre-MI period. Higher proportions of Chinese patients and both groups of controls had never smoked and fewer were current smokers than the respective groups of Japanese men. Physical activity was lower in the Chinese than Japanese men.

According to Stavig et al(419), estimates of prevalence of elevated blood pressure levels ( $B P>140 / 90 \mathrm{~mm} \mathrm{Hg}$ ) among Californian Chinese men, ages $18-49$ years, were slightly lower than for comparable white men ( $11.8 \%$ vs $15.0 \%$ ). For Chinese men, ages $50+$ years, the prevalence of elevated blood pressure by this criterion was higher than for white men ( $45.0 \%$ vs $38.5 \%$ ). Prevalence of elevated blood pressure levels for Chinese women compared to white women was reversed: they were slightly higher in the 18-49 year age group ( $6.4 \%$ vs $4.8 \%$ ) and slightly lower $34.3 \%$ vs $36.4 \%$ for women, ages 50 or more years. Levels of hypertension awareness among Chinese male and female hypertensives 50 years and older in California in 1979 were roughly comparable to those of their white male and female cohorts.

Data on percentages of hypertensive Chinese Americans under drug treatment show that more Chinese hypertensives in the younger age group (18-49) were in treatment than their white hypentensive male cohorts. No stable estimates are available for Chinese women in this age-group.

The data on smoking among Chinese Americans by gender and age as presented in Table 71, taken from Kumanyika and Savage (421), shows that fewer young Chinese men were smokers than young white men (26.6\% vs $34.6 \%$ ) but more older Chinese men were smokers than older white men. Regardless of age, however, a significantly larger percentage of white women were smokers than Chinese American women. In the case of those who described themselves as current or former smokers, however, white men and women were more likely to describe themselves as current or former smokers than were Chinese American men and women of all ages. The same results are obtained for numbers of cigarettes smoked. Regardless of age and gender, whites smoke more cigarettes on the average than Chinese Americans.

## D: Biologic and/or Physiologic Variables in Filipino Americans

There is a marked paucity of health information on Filipinos and other Asian groups. Other than the mortality data for Filipinos previously reviewed, the only other specific data - identified from the California Hypertension Survey on blood pressure and smoking are noted below.

## Hypertension


#### Abstract

* Hypertension is a significant public health problem in Filipino $\%$ * Americans. The same number or more Filipinos are receiving treat-* * ment for their hypertension compared to their white cohorts, yet $\#$ $\%$ fewer have achieved blood pressure control. In striking contrast $*$ * to other gender/ethnic groups, Asian and non-Asian, Filipino * * women, in particular, were more likely to have high blood pres- * * sure that was not under control. *


以
Mean blood pressure levels among Filipino men in California in 1979 were estimated from the California Hypertension Survey data. Stavig et al(419) note that the Filipino population of California tripled between 1970 and 1980. Estimates of elevated blood pressure prevalence ( $B P>140 / 90 \mathrm{~mm} \mathrm{Hg}$ ) among California Filipino men and women were higher at all ages and substantially higher than for whites in the age-sex groups with relatively higher prevalences. Among Filipino men, ages $18-49$ years, $29 \%$ were found to have elevated blood pressures as compared to $15.0 \%$ of whites. Among those Filipino men, ages $50+$ years, $50.8 \%$ had elevated blood pressures as compared to $38.5 \%$ of white male cohorts. Filipino women were similarly more likely to have high blood pressure than their white female cohorts. Filipino women were more likely to have elevated blood pressure at an older age, although Filipino men were more likely to have elevated blood pressure when they are younger.

## Smoking



* Filipinos, like their fellow Asians (at least those in Califor- * $\pm$ nia) do not show evidence of an excess prevalence of smoking com-* * pared to whites.
"

Data on the prevalence of smoking and pattern of smoking among younger and older Filipino men and women compared to whites were also available from Igra et al.(420) Like the Japanese and Chinese, fewer Filipino men and women in all age-groups were current regular smokers compared to whites ( $26.0 \%$ vs $63 \%$, respectively, among men and $14.3 \%$
vs $29.4 \%$ among women). Filipino men smoked 17.7 cigarettes on the average vs 27.4 cigarettes smoked by white men. Filipino women smoked 8.0 cigarettes vs 23.2 cigarettes smoked by white women.

Of those who were smokers, slightly more Filipino men than white men expressed a desire to quit smoking, though comparable or fewer Filipino women wanted to quit smoking than white women [Table 72].(421)

## I INTRODUCTION

The available epidemiological data on Native Americans are limited by many of the same deficits found with other minorities; namely, data on these groups are either aggregated in a "non-white" or "other" category, or Native Americans are treated as a single group with no distinctions made for tribal origins. Although many may debate the validity of these subgroup distinctions, given the relative homogeneity of social status across all tribes, there is ample evidence, nevertheless, of important health differences between Indian tribes. These differences have been attributed to differences in cultures, in level of acculturation, in degree of urbanization, and in degree of conflict between the native culture and the social standards of the contiguous American communities. $(500,501,502,503)$ Thus, some of the evidence reviewed here reflects estimates of the overall status of Native Americans and Alaska Eskimos, but almost certainly does not directly reflect the specific cardiovascular status of all tribes. In certain cases, the data are specific to one or more tribes, and caution should be taken in these cases not to overgeneralize these findings to other Native American tribes.

The census designation "Native American" includes American Indians, Eskimos, and Aleuts. Native Americans comprised $0.6 \%$ of the U.S. population in the 1980 census - 1,418,000 of the $226,505,000$ Americans counted.(504) This is an increase from the previous $0.4 \%$ of the U.S. population in the 1970 census. The Navajo, the largest of several hundred Native American tribes, numbered approximately 150,000 in the mid-1970s. (505) States with the largest Native American populations are Arizona, Oklahoma, California, New Mexico, and North Carolina, but the federally recognized Indian tribes are spread throughout more than 25 states. $(505,524)$

Prior to 1940, $90 \%$ of Indians lived on reservations, but by 1977 more than $50 \%$ lived in urban centers. Sievers and Fisher(506) point out that southwestern Indians have remained more isolated and less racially mixed than Indians in other regions. At the time of the Sievers and Fisher review(506) 6\% of southwestern Indians over age 15 versus $20 \%$ of this same population under age 15 are reported to have some non-Indian admixture. The Native American population is disproportionately poor, has a lower life expectancy than all other U.S. races, and is younger than the U.S. population as a whole (i.e. median age 18.4 vs 28.1 years for the U.S. population in the 1970 census). $(506,507)$

人

* Heart disease is a significant contributor to all-cause mortality*
* in Native Americans, but is proportionately less of a contributor*
* than in the general population, due apparently to the greater $\quad *$ $*$ contribution of noncardiovascular causes such as automobile * $\%$ accidents and chronic liver disease to all-cause mortality. When * * these are better controlled, it is likely that heart disease will* $\stackrel{+}{2}$ increase in prevalence, as Native Americans live longer. $\quad *$ *

In a recent report on cardiovascular disease in Native American populations, Kumanyika and Savage(508) reviewed the evidence on proportionate mortality from CHD compared to other major causes of death and noted that the mortality profile of Native Americans differs from the U.S. population, as well as from the other racial minorities. Although the majority of deaths are from heart disease in both the Indian and general populations, the proportionate mortality from heart disease in Native Americans is half that of the general population [Table 80].(509) By the same token, accidental death rates are nearly as high as those from heart disease and are more than three times as high as the proportionate mortality from accidents in the general population. A similar pattern of excess mortality from chronic liver disease is also evident in Native Americans [Table 80].(508)

Other competing causes of death in Native Americans such as infant mortality, mortality from tuberculosis, gastrointestinal disease, accidents, and alcoholism have decreased significantly since 1955, but remain disproportionately higher than in the general population, even as recently as 1980 [Table 81]. (508)

Mortality rates from cardiovascular diseases reported by NCHS for 1979-1981 include comparisons between Native Americans (treated as a single group), whites, Blacks, and Asians (treated as a single group). Data on Hispanic mortality were aggregated with data from whites. Therefore, comparisons between Native Americans and the other minority groups and whites conceal whatever differences in CHD mortality exist between these groups and Hispanics.

Native Americans show evidence of reduced heart disease mortality in men and in women compared to the comparable white populations. At all ages and for both men and women, mortality from heart disease is significantly lower than for Black American men and women, but may be slightly higher than for Asians [Table 82].

Kumanyika and Savage(508) compiled data to show changes over time in CHD mortality by age and region. Data on heart disease mortality in the available Indian Health Service tabulations for 1975 and 1979-1980 are limited in detail, but do show a marked decrease in heart, cerebrovascular, and atherosclerosis mortality in native Americans that was comparable to that observed in the general U.S.
population between 1970-1975. There was a substantially smaller decrease, however, in mortality from hypertension [Table 83].
However, the sharp increase in mortality due to suicide and cirrhosis of the liver raises the possibility that the reduced CHD mortality may have been due, at least in part, to competing causes of death rather than to a basic reduction in CHD risk.

Under age 35 years, the heart disease death rate for Native Americans is approximately twice as high as for all other ethnic groups. Above the age of 44 years, heart disease mortality increases less steeply with age in Native Americans than in the general population, and Native American rates are lower than those in all other groups for age-groups over 45 years. $(508,510)$ A later crossover in atherosclerosis and cerebrovascular disease death rates is also observed.
(ii) Morbidity
*
$\therefore$ Some preliminary data point to a pattern of increasing CHD in- $\quad *$

* cidence in certain urban Native Americans. However, other data
* for southwestern tribes indicate low prevalence of CHD despite
* high rates of obesity, of diabetes, and increasing hypertension $\star$ rates.
* 

Preliminary prevalence and incidence data suggest that coronary heart disease and stroke risk may be increasing substantially in this population, especially among those residing outside the southwestern states. $(506,511)$ Sievers and Fisher(506) report that CHD is a relatively uncommon problem in southwestern Indian tribes despite high rates of obesity, diabetes, and increasing rates of hypertension. They attribute these findings to low prevalence of major biologic and behavioral risk factors. However, the recent report by Gillum et al(511) on CHD risk factors in Native Americans in Minnesota and Montana (mainly Chippewa/Ojibwe people were studied) points to increases in both standard CHD risk factors and in the incidence and prevalence of cardiovascular diseases in urban American Indians. This pattern of increasing CHD is also beginning to be evident in the form of increasing rates of myocardial infarction in southwestern American Indians, especially among the Hopis. $(508,506)$

## II: EXPLANATIONS FOR DIFFERENCES



* In general, the data on CHD risk factor status for Native Amer- *
* icans are even more limited than that available for other minor- *
$*$ ity groups. Such data that do exist represent a particular tribe *
* or subgroup rather than the entire Native American population. *
* Cautious interpretation is therefore required. *

中

Kumanyika and Savage(508) noted that a large portion of the literature pertinent to cardiovascular risk factors reports on Indians in the Southwest and, in particular, on Pimas, who are reported to have the highest prevalence of type II diabetes in the United States. (506) This group appears to be the exception to the rule that a high prevalence of obesity is related to high cholesterol and a high prevalence of heart disease. (512) However, other tribes have been studied including the Papago, Navajo, Apache, Hopi, Ojibwe, Sioux and Winnebago in Minnesota, the Crow and northern Cheyenne in southwest Montana, the Arapaho and Shoshone in Wyoming, the Seminoles in Oklahoma and Florida, the Alaska Eskimos and Aleuts, and the Seneca in upstate New York. Nonetheless, these papers provide an incomplete picture of CHD risk for the subgroups listed and ignore a substantial portion of the Indian population.

It is also important to note that the history of the Native American population is unique and varies greatly between the Indian tribes. These factors have affected the amount and quality of the health and disease data available for this population. To further complicate the problem, many of these data are based on potentially biased data sources and on anecdotal material.

Explanations of the patterns of cardiovascular diseases observed need to consider not only genetic/biological factors, but cultural and economic factors as well. Native Americans, as do most Americans, differ in their degree of racial admixture, and there are significant inconsistencies in the classification of racially mixed Indians (i.e., they are classified as either Indians or as whites).

## A: Biologic and/or Physiologic Variables

Hypertension
*
$\star$ Hypertension appears to be an important health problem for Native ${ }^{*}$

* Americans, though apparently less so than for the white popula- * $*$ tion.
$\star$


The limited evidence available on the prevalence of hypertension in Native Americans does not permit us to draw confident conclusions about the significance of this risk factor for all Native Americans. Sievers (513) noted that, over the past 35 years, reports typically find lower prevalence of hypertension in Native Americans than in whites. (506) This difference was evident both in the southwest and in other regions. However, he also noted higher rates of high blood pressure among Indians who had migrated to urban centers, and higher blood pressures in Indian diabetics than in nondiabetics. However, these trends are not consistent for all Indian groups. For example, studies on White Mountain Apache men found a high prevalence of high blood pressure. On the other hand, no blood pressure differences were found between Seminoles and whites in Florida, despite significantly higher percentages of obese and diabetic Seminoles.

The results of a survey on a diverse but nonrandom sample of Navajos in Arizona and New Mexico found that Navajo men had higher resting blood pressures and a greater prevalence of hypertension than Navajo women, but they did not show the expected positive increase of blood pressure with age. Level of acculturation was not found to be associated with blood pressure.(514)

Gillum et al $(511,515)$ reported a survey of blood pressure and related CHD risk factors in all first-, second-, and third-grade children in Minneapolis public schools, and a survey of two adult Indian populations in Minneapolis (one from an Indian housing project and the other from community screenings during American Indian week). Both studies were mainly on Chippewa/Ojibwe Indians. The results from the children's survey found higher systolic pressures but lower diastolic pressures in the Native American children than in their white peers. Similar results were obtained in the survey of the adults. Blood pressure readings were comparable in both Native Americans and whites. These American Indians also reported a higher prevalence of diabetes, obesity, and smoking. Thus, though hypertension prevalence appears to be approximately equivalent in whites and in urban Ojibwe Indians in Minnesota, Ojibwe Indians have a higher associated risk profile due to a higher prevalence of obesity, diabetes, and smoking.

## Blood Lipids and Lipoproteins

* Serum cholesterol levels in some American Indians are lower than * $\star$ in the general population but the relationship of these lower * * levels to CHD incidence is not clear. *
* 

Kumanyika and Savage(508) note that comparisons of cholesterol levels between whites and Native Americans indicate equal or lower levels in Native Americans. Sievers(516) reported that, in a comparison of 746 southwestern Indians, 70 non-southwestern Indians, and 163 whites, both Indian groups had lower cholesterol levels than whites, showed no cholesterol level increases with age, and no differences in levels between men and women. Lower cholesterol levels were found in Pima and Papago Indians than in Apache and Navajo Indians, despite greater prevalence of obesity among the Pimas.

In a report of cholesterol levels in children, Savage et al found that Pima and white children had similar cholesterol levels at birth, but cholesterol levels in Pimas did not increase with age in adulthood.

Comparisons of diabetic and nondiabetic Pimas indicated slightly higher cholesterol levels in the diabetics, but all other relationships were consistent with previous data on cholesterol in Pimas. (517) Metabolic studies have suggested that there may be significant differences in apoprotein and lipoprotein metabolism in Pimas which might account for their different lipid profile. $(518,519,520)$

Diabetes and obesity are major public health problems in most Native American populations.

The Pima Indians have an unusually high prevalence of diabetes, but an excess of glucose intolerance prevalence appears to be typical of many adult Indian populations.(521)

## Cigarette Smoking and Alcohol Use

## 

* Although insufficient data are available to draw firm conclusions* * about trends in cigarette and alcohol use, or their contribution * * to CHD, the available data do suggest that prevalence of cigar- * * ette smoking is less consistent between Native Americans from * $\star$ different geographic subgroups, but that prevalence of alcohol *
$\%$ abuse is more consistent. Native American men have higher rates * $\% \quad$ of cigarette smoking and alcohol use than do the women. *


Sievers(516) documented cigarette and alcohol use patterns in American Indians in a report based on interviews of patients at the Phoenix PHS Hospital. He found that heavy cigarette smoking (i.e.>1 pack/day) was rare among southwestern Indians, that smoking habits of non-southwestern Indians were similar to those of the general population, and that Indian women outside the southwestern area were more likely to be heavy smokers. Heavy alcohol (i.e. >1.6 ounces of absolute alcohol, more than once a week) was most common in southwestern Indians and significantly greater in other groups of Indians than in whites. Further, that both heavy cigarette and alcohol use were more frequent among men than among women in all groups studied. This is consistent with the results of the review of alcoholism in American Indians by Brod and Thomas. (522)

A similar study by Porter et al(523) of students, under age 20 years, in Anchorage, Alaska found that Native American students were more likely than white students to be users of substances, and more likely than any other ethnic group students to have tried drugs in addition to alcohol and tobacco.

## B: Socioeconomic and Sociocultural Factors

Native Americans have a peculiar social, political, and cultural history in the United States. What is especially unusual about their history is the fact that though they share (with other minorities) a history of racism, Native Americans were not originally foreign to the United States. Rather, they continue to exist on some of the
same lands their ancestors once owned, but now they are "excluded minorities" on those same lands. At the very core of the social dynamics of modern Indian life is the ongoing struggle between their native cultural heritage and lifestyle and the continuing encroachment of acculturative pressures. This struggle has led to divisions within tribes between the generations, and shifts away from native lifestyle and habits towards increasing incorporation of western dietary and lifestyle habits.

Little reseach has been conducted on the contribution of socioeconomic factors such as low income, high unemployment, and low education on cardiovascular disease mortality, morbidity, and risk status in Native Americans.

Although some socioeconomic factors may contribute to an unfavorable risk profile, other sociocultural factors may operate to confer some protection against coronary heart disease. Many Native American tribes maintain low cholesterol diets, engage in strenuous physical activity as part of their daily lives, and have less time-pressure and lifestyles that display limited interpersonal competition. (521) Recent increases in urbanization, in smoking, in the fat content of diets, and in other behavioral risk factors associated with increased westernization in Native American youth in several tribes suggest the possible beginning of increased coronary heart disease risk in the population.

BLACK AMERICANS
Tables $1-36$. . . . . . . . . . . . . . . . . 96-131
(There are no tables 37-39)

Figures $1-19$. . . . . . . . . . . . . . . . . 132-150

HISPANIC AMERICANS

Tables 40-42 . . . . . . . . . . . . . . . . . 152-154
(There are no tables 43-49)

ASIAN/PACIFIC ISLANDER AMERICANS
Tables 50-72 . . . . . . . . . . . . . . . . . 156-184
(There are no tables 73-79)

NATIVE AMERICANS

Tables 80-83 . . . . . . . . . . . . . . . . . 186-190

## TABLE 1

Disability Days Attributed to the Atherosclerotic Diseases and Other Major Chronic Diseases, United States*

|  | Hospital <br> Days | Bed <br> Days | Wostricted <br> Activity |  |
| :--- | :---: | :---: | :---: | :---: |
| Disease |  |  | 66 | 21 |
| Days |  |  |  |  |

Source: Prepared by the National Heart, Lung, and Blood Institute; data from the National Center for Health Statistics.
"All data in this table are for 1978, except hospital days, which are for 1977. Categorles of disability days are not mutually exclusive.
**A numerical value is not cited because it was too small to meet standards of reliability or precision.

* " data unavallable.

From: Report of the Working Group on Arteriosclerosis of the National Heart, Lung, and Blood Institute, 1981 [Table 7, p.40]. Arteriosclerosis: 1981, Volume 1, NIH/PHS/DHHS. NIH Publication No.81-2034.

TABLE 2

Black/White Ratios of Death Rates for Coronary Heart Disease By Age and Sex, United States, 1980

| AGE | Coronary Heart Disease 410-414 | Acute <br> Myocardial Infarction 410 | $\begin{gathered} \text { Other } \\ \text { CHD } \\ 411-414 \end{gathered}$ |
| :---: | :---: | :---: | :---: |
| Male |  |  |  |
| Total (1) | 0.90 | 0.79 | 1.06 |
| 25-64 (1) | 1.11 | 0.94 | 1.49 |
| 35-74 (1) | 0.98 | 0.83 | 1.26 |
| 25-34 | 2.28 | 2.00 | 2.81 |
| 35-44 | 1.45 | 1.28 | 1.82 |
| 45-54 | 1.19 | 0.98 | 1.66 |
| 55-64 | 1.00 | 0.84 | 1.34 |
| 65-74 | 0.84 | 0.71 | 1.04 |
| 75-84 | 0.75 | 0.66 | 0.84 |
| 85+ | 0.64 | 0.64 | 0.63 |

Female

| Total (1) | 1.19 | 1.15 |  |
| :--- | :--- | :--- | :--- |
| $25-64$ (1) | 1.98 | 1.75 | 1.26 |
| $35-74$ (1) | 1.54 | 1.36 | 2.42 |
|  |  |  | 1.83 |
| $25-34$ | 3.30 | 3.28 |  |
| $35-44$ | 2.90 | 2.70 | 3.33 |
| $45-54$ | 2.28 | 1.02 | 3.27 |
| $55-64$ | 1.78 | 1.07 | 2.83 |
| $65-74$ | 1.22 | 0.84 | 1.46 |
| $75-84$ | 0.90 | 0.72 | 0.95 |
| $85+$ | 0.65 |  | 0.62 |

(1) Based on rates age-adjusted by the direct method to the U.S. population, 1940

[^2]TABLE 3

Proportionate Mortality for Coronary Heart Disease By Age, Color, and Sex; U.S., 1980

| AGE | MALES |  |  | FEMALES |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deaths All Causes | Deaths CHD | Percent CHD | Deaths All Causes | Deaths CHD | Percent CHD |
|  | Black |  |  |  |  |  |
| Total | 130,138 | 22,760 | 17.5 | 102,997 | 20,605 | 20.0 |
| 25-64 | 56,224 | 8,695 | 15.5 | 34,415 | 5,124 | 14.9 |
| 35-74 | 77,306 | 15,046 | 19.5 | 54,743 | 10,712 | 19.6 |
| 25-34 | 8,013 | 210 | 2.6 | 3,400 | 74 | 2.2 |
| 35-44 | 8,521 | 844 | 9.9 | 4,819 | 370 | 7.7 |
| 45-54 | 15,156 | 2,617 | 17.3 | 9,660 | 1,333 | 13.8 |
| 55-64 | 24,534 | 5,024 | 20.5 | 16,536 | 3,347 | 20.2 |
| 65-74 | 29,095 | 6,561 | 22.6 | 23,728 | 5,662 | 23.9 |
| 75-84 | 21,046 | 5,172 | 24.6 | 22,371 | 6,031 | 27.0 |
| 85+ | 8,534 | 2,273 | 26.6 | 13,115 | 3,767 | 28.7 |

White

| Total | 933,878 | 285,771 | 30.6 | 804,729 | 233,288 | 29.0 |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: |
| $25-64$ | 282,125 | 80,575 | 28.6 | 154,716 | 24,269 | 15.7 |
| $35-74$ | 500,826 | 164,231 | 32.8 | 308,643 | 71,595 | 23.2 |
|  |  |  |  |  |  |  |
| $25-34$ | 27,303 | 744 | 2.7 | 10,395 | 159 | 1.5 |
| $35-44$ | 28,344 | 5,189 | 18.3 | 15,520 | 966 | 6.2 |
| $45-54$ | 68,306 | 21,066 | 30.8 | 38,328 | 4,769 | 12.4 |
| $55-64$ | 158,172 | 53,576 | 33.9 | 90,473 | 18,365 | 20.3 |
| $65-74$ | 246,004 | 84,400 | 34.3 | 164,322 | 47,495 | 28.9 |
| $75-84$ | 229,619 | 78,931 | 34.4 | 240,748 | 83,194 | 34.6 |
| $85+$ | 118,549 | 41,727 | 35.2 | 215,691 | 78,265 | 36.3 |

From: Vital Statistics of the U.S., National Center for Realth Statistics

## TABLE 4

## Myths and Facts About CHD in U.S. Blacks

Myth Fact

CHD is uncommon in blacks.

Blacks rarely have myocardial infarction.

Blacks rarely have angina.

Whites have much more CHD than blacks in the United States.

Blacks are immune to CHD.

CHD is the leading cause of death in U.S. blacks.

Myocardial infarction hospitalization rates are high in blacks, with higher case fatality rates than for whites.

Angina occurs with high prevalence in U.S. blacks.

CHD mortality and prevalence rates are similar in black and white males. Black females have higher CHD mortality and prevalence rates than white females. Adequate data on incidence are lacking.

Blacks are relatively susceptible to CHD, but it is surprising that they do not have rates even higher than those observed.

From: "Coronary heart disease mortality in United States blacks, 19401978: Trends and unanswered questions," Gillum, R.F. and Liu, K.C. [Table 1]. American Heart Journal 108(3;2): 729, 1984

## TABLE 5

Prevalence of Definite and Suspect Coronary Heart Disease in United States Adults Ages 18 to 79 Years By Sex and Race: 1960-1962

| Manifestation | Number of adults in thousands |  |  |  |  |  | Rates per 100 adults |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Both sexes |  | Men |  | Women |  | Both sexes |  | Men |  | Women |  |
|  | White | Black | White | Black | White | Black | White | Black | White | Black | White | Black |
| All forms | 4948 | 586 | 2753 | 328 | 2195 | 258 | 5.1 | 5.1 | 5.9 | 6.3 | 4.3 | 4.2 |
| Definite |  |  |  |  |  |  |  |  |  |  |  |  |
| Total | 2832 | 293 | 1776 | 169 | 1055 | 124 | 2.9 | 2.6 | 3.8 | 3.2 | 2.1 | 2.0 |
| $\begin{array}{ccccccccccll}\begin{array}{c}\text { Myocardial } \\ \text { infarction* }\end{array} & 1305 & 116 & 926 & 89 & 379 & 27 & 1.3 & 1.0 & 2.0 & 1.7 & 0.7\end{array}$ |  |  |  |  |  |  |  |  |  |  |  |  |
| Angina pectoris | 1388 | 160 | 773 | 62 | 615 | 98 | 1.4 | 1.4 | 1.7 | 1.2 | 1.2 | 1.6 |
| Other ${ }^{\text {t }}$ | 139 | 17 | 77 | 18 | 61 | - | 0.1 | 0.2 | 0.2 | 0.3 | 0.1 | - |
| Suspect |  |  |  |  |  |  |  |  |  |  |  |  |
| Total | 2117 | 293 | 976 | 159 | 1140 | 134 | 2.2 | 2.6 | 2.1 | 3.1 | 2.2 | 2.2 |
| Angina pectoris | 2059 | 293 | 976 | 159 | 1083 | 134 | 2.1 | 2.6 | 2.1 | 3.1 | 2.1 | 2.2 |
| Other $\ddagger$ | 58 | - | - | - | 57 | - | 0.1 | - | - | - | 0.1 | - |

Source: Gordon T, Garst CC: Coronary heart disease in adults, United States 1960-1962. National Center for Health Statistics, Series 11. No. Iv. Washington, D.C., 1965, U.S. Government Printing Office.
*On electrocardiogram with or without angina pectoris or history of myocardial infarction.

+ Myocardial infarction history with myocardial infarction outside criteria or left ventricular ischemia on electrocardiogram.
$\ddagger$ Myocardial infarction history with electrocardiographic evidence of myocardial infarction or left ventricular ischemia.
Note: All categories exclusive, in descending priority.

[^3]
## TABLE 6

Prevalence Rates of Definite and Suspect Coronary Heart Disease in United States Adults By Age, Sex, and Race: 1960-62

| Age (yr) | Rates per 100 adults |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Total |  |  |  | Definite |  |  |  | Suspect |  |  |  |
|  | Men |  | Women |  | Men |  | Women |  | Men |  | Women |  |
|  | White | Black | White | Black | White | Black | White | Black | White | Black | White | Black |
| Total |  |  |  |  |  |  |  |  |  |  |  |  |
| 18 to 79 | 5.9 | 6.3 | 4.3 | 4.2 | 3.8 | 3.2 | 2.1 | 2.0 | 2.1 | 3.1 | 2.2 | 2.2 |
| 18 to 24 | - | - | - | - | - | - | - | - | - | - | - | - |
| 25 to 34 | 0.1 | 3.1 | 0.4 | - | 0.1 | 3.1 | 0.2 | - | - | - | 0.2 | - |
| 35 to 44 | 2.2 | 3.5 | 0.9 | 1.9 | 1.2 | - | 0.4 | 1.0 | 1.0 | 3.5 | 0.5 | 0.9 |
| 45 to 54 | 6.6 | 10.2 | 3.7 | 8.0 | 3.0 | 7.4 | 1.3 | 3.9 | 3.5 | 2.8 | 2.4 | 4.1 |
| 55 to 64 | 14.4 | 13.4 | 10.0 | 9.8 | 10.3 | 5.7 | 4.7 | 5.5 | 4.2 | 7.7 | 5.3 | 4.3 |
| 65 to 74 | 17.3 | 10.9 | 14.4 | 14.2 | 12.2 | 3.4 | 8.2 | 5.1 | 5.1 | 7.5 | 6.2 | 9.0 |
| 75 to 79 | 14.0 | - | 13.5 | - | 9.8 | - | 5.1 | - | 4.1 | - | 8.5 | - |

Source: Gordon T, Garst CC: Coronary heart disease in adults, United States 1960-1962. National Center for Health Statistics, Seriea 11, No. 10. Washington, D.C., 1965, U.S. Government Printing Office.

```
From: "Coronary Heart Disease in Black Populations: Mortality and
    Morbidity", Gillum, R.F. [Table IV]. American Heart Journal
    104(4;1):845,1982
```


## TABLE 7

Incidence or Hospitalization Rates for Acute Myocardial Infarction In U.S. Black and White Populations Per One Thousand Subjects

| Age/Sex |  | $\begin{aligned} & \text { Evans County,GA } \\ & 1960-1967 \end{aligned}$ | $\begin{aligned} & \text { Noehville } \\ & 1967-1968 \end{aligned}$ | $\begin{aligned} & \text { Baltimore } \\ & 1970-1972 \end{aligned}$ | $\begin{gathered} \text { Newark } \\ 1973 \end{gathered}$ | $\begin{gathered} \text { Columbio,SC } \\ 1968 \end{gathered}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 35-44 |  |  |  |  |  |  |
| Black | men | 1.79 | 1.02 |  | - | 0.84 |
| White | men | 8.28 | 1.84 |  | 0.783 | 31.25 |
| Black | women | ก 3.72 | 0.19 |  | 0.193 | 30.38 |
| White | women | ก 0.69 | 0.20 |  | 0.113 | 30.06 |
| 45-54 |  |  |  |  |  |  |
| Black | men | 5.79 | 1.55 | 0.6 | 1.16 | 3.97 |
| White | men | 10.48 | 5.50 | 1.9 | 1.09 | 5.76 |
| Black | women | ก 5.10 | 1.41 | 0.2 | 0.85 | 0.68 |
| White | women | ก 4.14 | 1.05 | 0.4 | 0.28 | 1.33 |
| 55-64 |  |  |  |  |  |  |
| Black | men | 1.52 | 3.47 | 0.8 | 2.41 | 7.61 |
| White | men | 17.79 | 9.82 | 2.9 | 3.38 | 10.75 |
| Black | women | ก 8.97 | 1.64 | 1.0 | 1.64 | 2.91 |
| White | women | ก 5.52 | 2.77 | 1.1 | 0.88 | 2.56 |
| 65-74 |  |  |  |  |  |  |
| Black | men | 8.69 | 3.87 |  | 4.61 | 6.27 |
| White | men | 32.14 | 12.75 |  | 5.37 | 18.82 |
| Black | women | n 6.21 | 1.77 |  | 2.18 | 5.75 |
| White | women | ก 19.72 | 6.49 |  | 1.31 | B.47 |

Source: RF Gillum, Ref 2

From: "Prevalence and Incidence of Ischemic Heart Disease in U.S. Black and White Populations", Henderson, M. and Savage, D.D. [Table 7]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

## TABLE 8

Estimated Rate（1）of Hospital Discharges（2）for Acute MI and Chronic CHD for White and Black Men and Women in Selected Age Groups； United States， 1981

| DJAGNOSIS（3） | AGE | MEN |  | UOMEN |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | WН】丁 | BLAC | 世НІ | blā̃ |
| TOTAL CHD | 25－44 | 2.8 | 2.1 | 0.7 | 1.3 |
|  | 45－64 | 26.6 | 13.2 | 9.7 | 9.5 |
|  | 65－74 | 43.6 | 18.6 | 25.0 | 18.1 |
|  | $75+$ | 51.6 | 24.2 | 43.9 | 26.7 |
| ACUTE MJ | 25－44 | 0.7 | 0.6 | 0.1 | 0.2 |
|  | 45－64 | 6.5 | 3.1 | 2.4 | 1.4 |
|  | 65－74 | 13.2 | 5.6 | 6.0 | 5.3 |
|  | $75+$ | 14．5 | 4.8 | J 0.2 | 5.2 |
| CHRONIC CHD | 25－44 | 2.1 | 1.5 | 0.6 | ］．1 |
|  | 45－64 | 20.1 | 10.1 | 7.3 | 8.1 |
|  | 65－74 | 30.4 | 13.1 | 19.0 | 12.8 |
|  | $75+$ | 37.1 | 19.4 | 33.7 | 21.5 |

（1）Per 1,000 population（civilian）．
（2）Discharged alive or dead．
（3）First－listed diagnosis（iCD／9 code）：
Coronary heart disease（410－414） Acute myotardial infarction（4］0） Other CHD（411－414）．

|  | Hypertensive ${ }^{1}$ |  |  | Never diagnosed ${ }^{2}$ |  |  | On medication |  |  | On medication and controlled ${ }^{3}$ |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1960-62 | 1974-76 | 1976-80 | 1960-62 | 1974-76 | 1976-80 | 1960-62 | 1974-76 | 1976-80 | 1960-62 | 1974-76 | 1976-80 |
|  | Percent of population ${ }^{4}$ |  |  | Percent of total with hypertension 1,4 |  |  |  |  |  |  |  |  |
| All people $25-74$ years ${ }^{5}$. . | 20.3 | 22.1 | 22.0 | 51.1 | 36.4 | 26.6 | 31.3 | 34.2 | 56.2 | 16.0 | 19.6 | 34.1 |
| White men . | 16.3 | 21.4 | 21.2 | 57.6 | 42.3 | 40.6 | 22.4 | 25.9 | 38.3 | 11.8 | 15.1 | 20.9 |
| White women | 20.4 | 19.6 | 20.0 | 43.9 | 29.7 | 25.2 | 38.2 | 48.5 | 58.6 | 21.9 | 28.1 | 40.3 |
| Black men . | 31.8 | 37.1 | 28.3 | 70.5 | 41.0 | 35.7 | 18.5 | *24.0 | 40.9 | 5.0 | *12.7 | 16.1 |
| Black women . . | 39.8 | 35.5 | 39.8 | 35.1 | 28.9 | 14.5 | 48.1 | 36.4 | 60.6 | 20.2 | *22.3 | 38.3 |
| Standard error of percent |  |  |  |  |  |  |  |  |  |  |  |  |
| All people 25-74 years ${ }^{5}$. | 0.83 | 1.26 | 0.68 | 1.66 | 1.70 | 1.53 | 1.62 | 2.21 | 1.99 | 1.65 | 1.49 | 2.02 |
| White men . . | 0.95 | 2.19 | 1.04 | 3.75 | 2.63 | 1.80 | 3.07 | 3.22 | 2.47 | 2.59 | 2.56 | 2.01 |
| White women | 1.07 | 1.14 | 0.66 | 2.77 | 2.08 | 1.97 | 2.24 | 3.61 | 2.40 | 2.24 | 2.93 | 2.99 |
| Black men . . | 3.37 | 5.94 | 1.86 | 7.07 | 10.38 | 4.27 | 5.53 | 10.79 | 4.52 | 2.18 | 6.69 | 3.72 |
| Black women . . . . . . . . | 3.73 | 3.60 | 1.96 | 3.72 | 7.42 | 2.73 | 3.87 | 8.30 | 3.22 | 3.21 | 7.93 | 4.35 |

[^4]
## TABLE 10

Age-Adjusted Death Rates Due to Cerebrovascular Disease (430-438) 1981 - United States By Race and Sex

|  | Number of Deaths | Rate/100,000 | Ratio to <br> White Women |
| :--- | :---: | :---: | :---: |
| Total | 163,504 | 38.1 | 1.1 |
| Men | 66,429 | 41.7 | 1.3 |
| Women | 97,075 | 35.4 | 1.1 |
| White Men | 57,000 | 38.9 | 1.2 |
| White Women | 85,765 | 33.1 | 1.0 |
| All Other Men | 9,429 | 65.6 | 2.0 |
| All Other Women | 11,310 | 53.2 | 1.6 |
| Black Men | 8,760 | 72.7 | 2.2 |
| Black Women | 10,656 | 58.1 | 1.8 |

From: "Stroke Report," Kuller, L. [Table 1]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

TABLE 11
Ratio of Stroke Mortality By Age, 1980: Black and White

|  | Black Men/ <br> White Men | Black Women/ <br> White Women |
| :--- | :---: | :---: |
| $25-34$ | 3.5 | 3.5 |
| $35-44$ | 4.5 | 3.2 |
| $45-54$ | 3.8 | 3.3 |
| $55-64$ | 3.0 | 2.8 |
| $65-74$ | 2.0 | 2.1 |
| $75-84$ | 1.1 | 1.3 |
| $85+$ | 0.8 | 0.8 |
| Total | 1.8 | 1.8 |

[^5] the DHHS Task Force on Black and Minority Health, 1984-85

TABLE 12
Age-Adjusted Stroke Death Rates By Geographic Area 1978 Per 100,000 Ages 35 to 74 Years

|  | White <br> Men | White <br> Women | Black <br> Men | Black <br> Women |
| :--- | :---: | :---: | :---: | :---: |
| Colorado | 43 | 38 |  |  |
| Kansas | 50 | 43 |  |  |
| Utah | 38 | 47 |  | 75 |
| New York | 48 | 39 | 91 | 73 |
| Maryland | 47 | 38 | 115 | 173 |
| South Carolina | 79 | 54 | 231 | 158 |
| Georgia | 82 | 59 | 283 | 103 |
| Mississippi | 77 | 45 | 169 | 197 |

From: "Stroke Report," Kuller, L. [Table 7]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

## TABLE 13

Trends of Mortality Rates from Cerebrovascular Diseases (ICD 430-438), Age-Adjusted, Persons Aged 35 to 74 Years By Sex and Color, United States, 1968-78

| Year | White Men | White Women | Nonwhite Men | Nonwhite Women | A11 |
| :---: | :---: | :---: | :---: | :---: | :---: |
| 1968 | 155.4 | 110.8 | 340.2 | 292.1 | 148.1 |
| 1969 | 150.3 | 106.8 | 317.1 | 274.3 | 142.2 |
| 1970 | 146.8 | 105.8 | 304.0 | 261.9 | 139.3 |
| 1971 | 143.8 | 100.7 | 292.9 | 248.6 | 134.2 |
| 1972 | 144.8 | 99.7 | 280.4 | 249.8 | 133.6 |
| 1973 | 137.4 | 96.8 | 282.0 | 239.8 | 128.7 |
| 1974 | 129.4 | 91.3 | 260.8 | 216.6 | 120.7 |
| 1975 | 117.8 | 83.7 | 233.7 | 190.1 | 109.7 |
| 1976 | 108.9 | 78.5 | 216.6 | 176.9 | 102.2 |
| 1977 | 100.9 | 72.4 | 202.2 | 162.4 | 94.6 |
| 1978 | 93.9 | 68.7 | 194.7 | 148.6 | 88.7 |
| Change 1968-78 | -62.1 | -42.1 | -145.5 | -143.5 | -59.4 |
| Percent Change | -40.0 | -38.0 | -42.8 | -49.1 | -40.1 |
| Slope 1968-73 | -0.0174 | -0.0190 | -0.0348 | -0.0359 | -0.0288 |
| Standard Error | 0.0035 | 0.0036 | 0.0049 | 0.0048 | 0.0032 |
| Slope 1973-78 | -0.0789 | -0.0707 | -0.0769 | -0.0951 | -0.0761 |
| Standard Error | 0.0017 | 0.0021 | 0.0054 | 0.0041 | 0.0020 |

Report of the Working Group on Arteriosclerosis of the National Heart, Lung and Blood Institute. Arteriosclerosis 1981. Volume 2.

From: "Stroke Report", Kuller, L. [Table 25]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

## TABLE 14

Trends in Stroke Mortality 1974-1978 By State: In Percent, Annual Change, Age-Adjusted (35-74 Years)

|  | White <br> Women | White <br> Men | Black <br> Women | Black <br> Men |
| :--- | :---: | :---: | :---: | :---: |
| Colorado | 6.6 | 6.7 |  |  |
| Connecticut | 9.2 | 6.0 |  | 18.2 |

From: "Stroke Report," Kuller, L. [Table 28]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

TABLE 15

Percentage Decline in Stroke Mortality By Age, Race, and Sex, 1970-80

| WM | WW | BM | BW |  |
| :---: | :---: | :---: | :---: | :---: |
| $35-44$ | 44.9 | 41.7 | 44.6 | 56.0 |
| $45-54$ | 39.0 | 38.7 | 39.8 | 48.2 |
| $55-64$ | 47.6 | 37.6 | 44.8 | 48.9 |
| $65-74$ | 42.9 | 43.0 | 39.3 | 46.2 |
| $75-84$ | 37.2 | 37.9 | 26.0 | 31.3 |

Health United States, 1983.

From: "Stroke Report", Kuller, L. [Table 29]. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85

TABLE 16

## End-Stage Renal Disease

Prevalence By Primary Diagnosis and Race in Dialysis and Transplant Patients, 1982

| Prevalence, 1982 | White | Black | Other |
| :---: | :---: | :---: | :---: |
| Dialysis Patients |  |  |  |
| Primary Diagnosis (3 Leading) |  |  |  |
| 1. Nephritis/Nephrosis | 7,499 (20.6\%) | 2,130 (12.6\%) | 409 (20.8\%) |
| 2. Hypertension | 4,347 (11.9\%) | 4,687 (27.7\%) | 205 (10.4\%) |
| 3. Diabetes | 4,318 (11.8\%) | 1,884 (11.1\%) | 290 (14.8\%) |
| Total | 36,475 (66\%) | 16,938 (30\%) | 1,963 (4\%) |
| Transplant Patients |  |  |  |
| Primary Diagnosis (3 Leading) |  |  |  |
| 1. Nephritis/Nephrosis | 820 (24.8\%) | 158 (19.2\%) | 43 (28.7\%) |
| 2. Hypertension | 178 (5.4\%) | 198 (24.1\%) | 15 (10.0\%) |
| 3. Diabetes | 466 (14.1\%) | 33 (4.0\%) | 9 (6.0\%) |
| Total | 3,302 (77\%) | 822 (19\%) | 150 (4\%) |

From: ESRD Systems Branch, Health Care Financing Administration, 1984

TABLE 17
Multivariate Association of Risk Indicators With Time to Death and 20-Year Cumulative Risk of Death Attributed to IHD in Black Males Aged 40 to 64 Years in Evans County*

|  | Proportional <br> hazard <br> coefficients (P) | Logistic risk <br> functions <br> coefficients (p) |
| :--- | ---: | ---: |
| Intercept | $0.101(0.002)$ | $-12.128(0.14)$ |
| Age | $0.086(0.01)$ |  |
| SBP | $0.023(0.0000)$ | $0.018(0.01)$ |
| Cholesterol | $-0.066(0.06)$ | $-0.081(0.05)$ |
| Cholesterol $/ 100$ | $0.013(0.10)$ | $0.016(0.09)$ |
| Smoking current | $1.406(0.007)$ | $1.209(0.03)$ |
| Smoking past | $-0.092(0.93)$ | $-0.125(0.91)$ |
| Quetelet index | $2.726(0.42)$ | $5.305(0.19)$ |
| Quetelet index 2 | $-0.295(0.49)$ | $-0.614(0.19)$ |
| X2 (8 df) | 38 | 26 |
| P | 0.000 | 0.001 |

*Number with IHD: 31; number of examinees: 294.

From: "Ischemic heart disease rísk factors and twenty-year mortality in middle-age Evans County black males," Tyroler, H.A. et al. [Table VI]. American Heart Journal 108(3;2):745, 1984

| Cause of death (ICD-9) | Black males |  |  | White males |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | No. | Rate/1000 | Percent* | No. | Rate/1000 | Percent* |
| Total with death certificates | 450 | 19.2 | 100.0 | 4602 | 14.1 | 100.0 |
| All cardiovascular diseases | 203 | 8.6 | 45.1 | 2226 | 6.8 | 48.4 |
| Cerebrovascular diseases (430-438) | 30 | 1.3 | 6.7 | 152 | 0.5 | 3.3 |
| Myocardial infarction (410) | 78 | 3.3 | 17.3 | 1225 | 3.8 | 26.6 |
| Other ischemic heart disease (411-414) | 29 | 1.2 | 6.4 | 483 | 1.5 | 10.5 |
| Hypertensive heart disease (402) | 17 | 0.7 | 3.8 | 26 | 0.1 | 0.6 |
| Other hypertensive disease (401, 403-405) | 1 | 0.0 | 0.2 | 7 | 0.0 | 0.1 |
| Other cardiovascular disease | 48 | 2.0 | 10.7 | 333 | 1.0 | 7.2 |
| (390-459 exclusive of above) | 247 | 10.5 | 54.9 | 2376 | 7.3 | 51.6 |
| All noncardiovascular diseases | 0 | 0.0 | 0.0 | 14 | 0.0 | 0.3 |
| Genitourinary diseases (580-629) | 5 | 0.2 | 1.1 | 37 | 0.1 | 0.8 |
| Diabetes mellitus (250) | 128 | 5.4 | 28.4 | 1440 | 4.4 | 31.3 |
| Neoplastic diseases (140-239) | 15 | 0.6 | 3.3 | 155 | 0.5 | 3.4 |
| Gastrointestinal diseases (520-579) | 12 | 0.5 | 2.7 | 124 | 0.4 | 2.7 |
| Respiratory diseases (460-519) | 4 | 0.2 | 0.9 | 16 | 0.0 | 0.3 |
| Infectious diseases (001-139) | 63 | 2.7 | 14.0 | 455 | 1.4 | 9.9 |
| Accidents, sulcides, and homicides (800-999) Other disease | 20 | 0.9 | 4.4 | 135 | 0.4 | 2.9 |

Number of Deaths By Cause for Black and White Males Screened
For the Multiple Risk Factor Intervention Trial
*Percent of total number of deaths in racial group.

[^6]
## TABLE 19

 Comparison of Logistic Regression Coefficients(*) for Diastolic Blood Pressure for All-Cause and Cause-Specific Mortality For Black and ltiple Risk Factor Intervention TrialScreenee Cohort

|  | PC | Black |  | White |  | Difference in Coeff | SE |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Coeff | SE | Coeff | SE |  |  |
| All-cause mortality | $1 \dagger$ | 0.0195 | 0.0033 | 0.0170 | 0.0013 | 0.0025 | 0.0035 |
|  | $2 \ddagger$ | 0.0202 | 0.0035 | 0.0171 | 0.0013 | 0.0031 | 0.0037 |
| CVD death | 1 | 0.0261 | 0.0051 | 0.0301 | 0.0019 | -0.0040 | 0.0054 |
|  | 2 | 0.0299 | 0.0055 | 0.0322 | 0.0020 | -0.0023 | 0.0058 |
| CHD death | 1 | 0.0188 | 0.0072 | 0.0263 | 0.0021 | -0.0075 | 0.0075 |
|  | 2 | 0.0244 | 0.0078 | 0.0289 | 0.0024 | -0.0045 | 0.0082 |
| Death from cerebrovascular disease | 1 | 0.0623 | 0.0105 | 0.0372 | 0.0068 | 0.0251 § | 0.0125 |
|  | 2 | 0.0624 | 0.0109 | 0.0324 | 0.0072 | 0.0030 § | 0.0131 |

$P C=$ Participant category; Coeff = coefficient; $S E=$ standard error. *Estimated for fixed age, serum cholesterol, and cigarettes per day. tCategory 1 includes all participants in racial group.
\#Category 2 excludes those participants who reported previous hospitalization for a heart attack or who were taking medication for diabetes. $\S_{\mathrm{p}}<0.05$.

[^7]table 20
HDL Cholesterol By Age, Black-White Differences(*)

 $\begin{array}{ll}\text { From: } & \text { "High-density lipoprotein cholesterol in blacks and whites: Potential } \\ & \text { ramifications for coronary heart disease," Glueck, C.J. et al.[Table I]. } \\ & \text { American Heart Journal } 108(3 ; 2): 817,1984\end{array}$

Cigarette Smoking Status and Cigarettes Smoked Per Day for Black Persons, 25 to 74 Years 01d, By Sex and Age:
United States, 1971-1975 and 1976-1980( + )

| Sex, age (year) | Current smoker $\dagger$ |  |  |  | Smoking 25 or more cigarettes/day |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1971-1975 |  | 1976-1980 |  | 1971-1975 |  | 1976-1980 |  |
|  | Rate | SE | Rate | SE | Rate | SE | Rate | SE |
|  |  |  |  | P | Popul |  |  |  |
| Males: age-adjusted |  |  |  |  |  |  |  |  |
| rate§ ( $25-74$ years) | 55.4 | 3.7 | 50.7 | 1.9 | 7.5 | 1.9 | 8.5 | 1.8 |
| 25-34 | 72.1 | 7.4 | 61.0 | 3.4 | 12.3 | 4.7 | 5.8 | 2.4 |
| 35-44 | 52.7 | 9.7 | 50.0 | 7.2 | 6.3 | 3.7 | 14.1 | 5.4 |
| 45-54 | 52.8 | 6.7 | 52.1 | 6.1 | 8.5 | 4.2 | 7.8 | 2.3 |
| 55-64 | 45.9 | 6.8 | 50.1 | 4.6 | 4.4 | 2.9 | 11.0 | 3.2 |
| 65-74 | 39.4 | 8.3 | 26.9 | 4.5 | 1.6 | 1.0 | 2.8 | 2.2 |
| Females: age-adjusted |  |  |  |  |  |  |  |  |
| rate§ (25-74 years) | 46.2 | 3.0 | 31.6 | 1.8 | 3.0 | 1.1 | 3.9 | 1.2 |
| 25-34 | 62.7 | 5.3 | 35.6 | 3.1 | 7.6 | 3.1 | 4.7 | 2.3 |
| 35-44 | 57.4 | 6.9 | 40.7 | 6.8 | 3.8 | 2.5 | 8.0 | 3.1 |
| 45-54 | 49.8 | 6.8 | 34.8 | 4.0 |  |  | 3.2 | 2.3 |
| 55-64 | 21.7 | 5.4 | 24.1 | 5.2 |  |  | 0.3 | 0.3 |
| 65-74 | 19.1 | 5.4 | 12.8 | 2.4 | - | - | 1.2 | 1.2 | SE = Standard error; $=$ data not available (quantity zero).

*Data from the National Health and Nutrition Examination Survey, Division of Health Examination Statistics,
National Center for Health Statistics, Hyattsville, Maryland.
$\dagger$ current smoker is a person who has smoked at least 100 cigarettes and who now smokes; includes SE = Standard error; $=$ data not available (quantity zero).

* Data from the National Health and Nutrition Examination Survey, Division of Health Examination Statistics,
National Center for Health Statistics, Hyattsville, Maryland.
tA current smoker is a person who has smoked at least 100 cigarettes and who now smokes; includes SE = Standard error; $=$ data not available (quantity zero).
*Data from the National Health and Nutrition Examination Survey, Division of Health Examination Statistics,
National Center for Health Statistics, Hyattsville, Maryland.
tA current smoker is a person who has smoked at least 100 cigarettes and who now smokes; includes
t current smoker is a person who has smoked at least 100 cigarettes and who now smokes; includes
$\ddagger$ Base of percent excludes persons with unknown smoking status.
§Age adjusted by direct method to the total U.S. population as estimated at the midpoint of the $1976-1980$
From: "Coronary heart disease risk factor trends in blacks between the first and second National Health and Nutrition Examination Surveys, United States, 1971-1980," Rowland, M.L. and Fulwood, R. [Table III]. American Heart Journal 108(3;2):774, 1984


## TABLE 22

## Percent Distribution of Adults Ages 35 to 64 Years By Cigarettes Smoked Per Day: United States 1965 and 1976(\%)

| Age (yr) | $1965$ <br> Cigarettes smoked per day |  |  |  | $1976$ <br> Cigarettes smoked per day |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Nonet | $<15 \ddagger$ | 15 to $24 \ddagger$ | 25 or more $\ddagger$ | Nonet | $<15 \ddagger$ | 15 to $24 \ddagger$ | 25 or more $\ddagger$ |
| White men |  |  |  |  |  |  |  |  |
| 35 to 44 | 43.3 | 12.1 | 25.4 | 19.2 | 53.5 | 8.0 | 18.8 | 19.7 |
| 45 11. 54 | 45.3 | 11.8 | 25.3 | 17.6 | 57.3 | 6.5 | 17.7 | 18.5 |
| 55 to 64 | 54.9 | 13.3 | 19.8 | 11.9 | 62.2 | 6.7 | 17.3 | 13.9 |
| Whit women |  |  |  |  |  |  |  |  |
| 35 t1 44 | 56.1 | 15.9 | 19.9 | 8.1 | 61.9 | 11.3 | 17.3 | 9.6 |
| 45 to 54 | 61.8 | 15.9 | 16.5 | 5.7 | 61.8 | 11.0 | 17.6 | 9.6 |
| 55 to 64 | 74.3 | 11.3 | 11.0 | 3.5 | 69.3 | 11.2 | 13.4 | 6.1 |
| Black men |  |  |  |  |  |  |  |  |
| $35 \text { to } 44$ | 32.7 | 28.6 | 30.6 | 8.1 | 41.1 | 22.6 | 26.4 | 9.8 |
| 45 to 54 | 37.6 | 25.3 | 30.6 | 6.6 | 43.3 | 19.5 | 27.4 | 9.8 |
| 55 t ) 64 | 48.2 | 30.0 | 17.9 | 3.9 | 59.5 | 15.6 | 22.3 | 2.5 |
| Black women |  |  |  |  |  |  |  |  |
| 35 to 44 | 57.1 | 27.2 | 13.0 | 2.6 | 58.7 | 24.9 | 15.7 | 0.6 |
| 45 to 54 | 67.8 | 21.5 | 9.6 | 1.2 | 63.4 | 16.5 | 16.5 | 3.7 |
| 55 to 64 | 83.5 | 12.8 | 3.2 | 0.5 | 59.9 | 25.3 | 10.7 | 4.2 |

- Data from Kleinman JC. Feldman JJ, Monk MA: The effects of changes in smoking habits on coronary heart disease mortality. Am J Public Health 69:745, 1979. By permission.
${ }^{+}$Excludes respondents with current smoking status unknown.
$\ddagger$ Excludes respondents with number of cigarettes smoked unknown.
Source: Heaith Interview Surveys 1965 and 1976.

From: "Coronary heart disease in black populations II. Risk factors", Gillum, R.F. and Grant, C.T. [Table III]. American Heart Journal 104(4;1):855, 1982
TABLE 23

|  | PC | Black |  | White |  | Difference in coeff | SE |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Coeff | SE | Coeff | SE |  |  |
| All-cause mortality | $1 \dagger$ | 0.0244 | 0.0032 | 0.0226 | 0.0008 | 0.0018 | 0.0033 |
|  | $2 \dagger$ | 0.0261 | 0.0034 | 0.0239 | 0.0008 | 0.0022 | 0.0035 |
| CVD death | 1 | 0.0256 | 0.0051 | 0.0223 | 0.0012 | 0.0033 | 0.0052 |
|  | 2 | 0.0293 | 0.0054 | 0.0248 | 0.0012 | 0.0045 | 0.0055 |
| CHD death | 1 | 0.0309 | 0.0067 | 0.0222 | 0.0013 | 0.0087 | 0.0068 |
|  | 2 | 0.0324 | 0.0074 | 0.0250 | 0.0014 | 0.0074 | 0.0075 |
| Death from cerebrovascular disease | 1 | 0.0103 | 0.0147 | 0.0260 | 0.0042 | -0.0157 | 0.0153 |
|  | 2 | 0.0146 | 0.0147 | 0.0287 | 0.0044 | -0.0141 | 0.0153 |
| $P C=$ Participant category; Coeff $=$ coefficient; $S E=$ standard error. <br> *Estimated for fixed age, diastolic blood pressure, and serum cholesterol. <br> $\dagger$ Category 1 includes all participants in racial group. <br> ${ }_{\dagger}$ Category 2 excludes those participants who reported previous hospitalization for a heart or who were taking medication for diabetes. |  |  |  |  |  |  |  |

Prevalence of Previously Diagnosed and of Undiagnosed Diabetes in the United States' Population Ages 20-74 Years, NHANES II, 1976-1980

| Race and Sex | Age: $20-74$ | 20-44 | 45-54 | 55-64 | 65-74 |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | Percent of Population (Standard Error) |  |  |  |  |
| Medical history of diabetes* |  |  |  |  |  |
| All races |  |  |  |  |  |
| Both sexes | 3.4 (.14) | 1.1 (.11) | 4.3 (.53) | 6.6 (.66) | 9.3 (.45) |
| Male | 2.9 (.25) | .6(.12) | 4.3 (.32) | 5.6 (.64) | 9.7 (.71) |
| Female | 3.8 (.24) | 1.5 (.22) | 4.3 (.67) | 7.4 (1.10) | 8.9 (.56) |
| White |  |  |  |  |  |
| Both sexes | 3.2 (.16) | 1.0 (.12) | 4.2 (.55) | 6.0 (.58) | 8.9 (.49) |
| Male | 2.8 (.27) | . 5 (.15) | 4.5 (.92) | 5.3 (.66) | 9.1 (.78) |
| Female | 3.6 (.23) | $1.4(.22)$ | 3.9 (.60) | 6.6(.91) | 8.8 (.64) |
| Black |  |  |  |  |  |
| Both sexes | 5.2 (.49) | 2.2 (.58) | 5.7 (1.46) | 13.1 (2.65) | 13.6 (1.35) |
| Male | 4.5 (.60) | 1.8 (.63) | 3.6 (1.48) | 9.2 (2.55) | 17.2 (2.87) |
| Female | $5.9(.99)$ | 2.6 (1.00) | 7.5 (2.33) | 16.3 (4.03) | 10.8(1.51) |
| Undiagnosed diabetes - NDDG Criteria** |  |  |  |  |  |
| All races |  |  |  |  |  |
| Both sexes | 3.2 (.35) | 0.9 (.31) | 4.2 (.81) | 6.2 (1.03) | 8.4 (.85) |
| Male | 2.8 (.41) | 0.8 (.39) | 3.6 (1.28) | 4.0 (1.03) | 9.5 (1.62) |
| Female | 3.6 (.42) | 1.0(.38) | 4.7 (1.14) | 8.1 (1.68) | 7.6 (.89) |
| White |  |  |  |  |  |
| Both sexes | 3.0 (.38) | 0.7 (.31) | 4.0 (.90) | 5.9 (1.24) | 8.0 (.85) |
| Male | 2.5 (.36) | 0.5 (.27) | 3.2 (1.25) | 3.8 (1.00) | 9.0 (1.38) |
| Female | $3.4(.52)$ | 0.8 (.40) | 4.6 (1.25) | 7.9 (2.08) | 7.3 (.95) |
| Black |  |  |  |  |  |
| Both sexes | 4.4 (.91) | 0.9 (.68) | 7.2 (3.05) | 7.7 (3.75) | 12.3(3.94) |
| Male | 4.0 (1.72) | 1.0 (.98) | 7.5 (6.40) | 5.2 (3.94) | 12.2 (7.23) |
| Female | 4.6 (1.35) | 0.9 (.91) | 7.0 (3.70) | 9.1 (5.92) | 12.3 (4.50) |

Undlagnosed diabetes - WHO Criteria**

| All races |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Both sexes | 3.4 (.35) | 0.9 (.35) | 4.2 (.81) | 6.8 (1.11) | 9.4 (.89) |
| Male | 3.0 (.41) | 0.8 (.48) | 3.6 (1.26) | 4.3 (1.06) | 10.4 (1.60) |
| Female | 3.9 (.43) | $1.0(.41)$ | 4.8 (1.15) | $9.0(1.76)$ | 8.5 (.93) |
| White |  |  |  |  |  |
| Both sexes | 3.2 (.38) | 0.7 (.35) | 4.0 (.91) | 6.5 (1.31) | 9.0 (.89) |
| Male | 2.7 (.36) | 0.5 (.42) | 3.3 (1.25) | 4.1 (1.03) | 10.0(1.57) |
| Female | 3.7 (.53) | 0.8 (.42) | 4.8 (1.26) | 8.6 (2.14) | 8.2 (1.09) |
| Black |  |  |  |  |  |
| Both sexes | 4.7 (.99) | 1.0 (.69) | 7.2 (3.05) | 9.4 (4.29) | 12.8(4.33) |
| Male | 4.1 (1.74) | 1.0 (1.01) | 7.5 (6.40) | 5.4 (3.98) | 12.2 (7.25) |
| Female | 5.1 (1.37) | 0.9 (.93) | 7.1 (3.71) | 11.6 (6.96) | 13.3 (4.99) |

*Based on a self-report that the person had been told by a doctor that he or she had diabetes, plus current or past use of diabetic therapy.
**Based on the results of a 75 gram oral glucose tolerance test conducted in the morning after an overnight $10-16$ hour fast in persons with no medical history of diabetes.

Source: National Center for Health Statistics. Hadden W. Harris M. Diabetes and Glucose Intolerance in Adults, 20-74 Years of Age, United States, 1976-80. Washington, DC. U.S. Government Printing Office (forthcoming). Vital and Health Statistics Series ll, data from the National Health Survey

[^8]Black Americans
TABLE 25
Five-Year Death Rates for the Multiple Risk Factor Intervention Trial Screenees(*) By Diabetes and Race

|  | N | CHD |  |  | CVD |  |  | All Cause |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | No. deaths | Deaths per 1,000 | $\begin{gathered} \text { Age** } \\ \text { adjusted } \end{gathered}$ | No. deaths | $\begin{gathered} \text { Deaths } \\ \text { per } 1,000 \\ \hline \end{gathered}$ | $\begin{gathered} \text { Age** } \\ \text { adjusted } \end{gathered}$ |  | $\begin{gathered} \text { Deaths } \\ \text { per } 1,000 \\ \hline \end{gathered}$ | $\begin{gathered} \text { Age** } \\ \text { adjusted } \\ \hline \end{gathered}$ |
| Diabetics | 5,245 | 92 | 17.5 | 13.5 | 128 | 24.4 | 18.9 | 265 | 50.5 | 42.3 |
| Black | 717 | 7 | 9.8 | 8.5 | 13 | 18.1 | 19.7 | 38 | 53.0 | 54.4 |
| Non-black | 4,528 | 85 | 18.8 | 14.2 | 115 | 25.4 | 18.4 | 227 | 50.1 | 40.1 |
| Non-Diabetics | 350,977 | 1,498 | 4.3 | 4.3 | 2,038 | 5.8 | 5.9 | 5,283 | 15.1 | 15.2 |
| Black | 22,444 | 86 | 3.8 | 4.1 | 170 | 7.6 | 8.1 | 465 | 20.7 | 21.8 |
| Non-black | 328,533 | 1,412 | 4.3 | 4.3 | 1,868 | 5.7 | 5.7 | 4,818 | 14.7 | 14.7 |

${ }^{*} \mathrm{~N}=356,222$; excludes men with history of MI.
**Adjusted to age distribution of all MRFIT Screenees.
From: "Diabetes and Risk of Coronary, Cardiovascular, and All Causes Mortality: Findings for 356,000 Men Screened by the
Multiple Risk Factor Intervention Trial (MRFIT). Stamler J, Wentworth D, Neaton J, Schoenberger JA, Feigal D, for the MRFIT Research Group. Circulation 70(Suppl. 2):II-161, 1984 (Abstract)

## TABLE 26

HDFP: Five-Year Mortality(a) By Race, Education, and Presence of LVH (b) at Baseline For All HDFP Stratum I Participants and Those Not on Medication at Baseline - Referred-Care Males, Ages 40 to 69 Years, Entry DBP 90 to 104 mm Hg

| Race/education | LVH- |  | LVH+ |  | \% Mortality |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Crude | Age-adjusted |  |
|  | (Deaths) | $N$ |  |  | (Deaths) | $N$ | LVH- | LVH+ | LVH- | LVH+ |
| All stratum I participants |  |  |  |  |  |  |  |  |
| WM $>$ HS | (18) | 446 | ( 1) | 8 | 4.0 | 12.5 | 4.5 | 10.2 |
| $W M=H S$ | (26) | 402 | ( 2) | 10 | 6.5 | 20.0 | 7.0 | 21.9 |
| WM < HS | (42) | 335 | ( 7) | 16 | 12.5 | 43.8 | 10.6 | 28.6 |
| BM < HS | (56) | 344 | (11) | 32 | 16.3 | 34.4 | 15.4 | 32.3 |
| Participants not on medication at baseline |  |  |  |  |  |  |  |  |
| WM $>$ HiS | (i3) | 347 | ( 1) | 8 | 3.8 | 12.5 | 4.4 | 10.2 |
| $W M=H S$ | (21) | 315 | ( 0 ) | 7 | 6.7 | 0.0 | 7.4 | 0.0 |
| WM < HS | (22) | 260 | ( 5) | 12 | 8.5 | 41.7 | 7.2 | 12.4 |
| $\mathrm{BM}<\mathrm{HS}$ | (41) | 268 | ( 6) | 18 | 15.3 | 33.3 | 14.9 | 33.2 |

"Age-adjusted by direct method. Standard population is age decade distribution of all white men ages 40 to 69 years with entry DBP 90 to 104 mmHg .

Geft ventricular hypertrophy determined by EKG. LVH + defined as major LVH by Minnesota Code; LVH - defined as all others.

[^9]
## TABLE 27

Mean and (SE) Nutrient Intake Per Day: Protein, Fat, Carbohydrate, Saturated Fat, Oleic Acid, and Linoleic Acid (gm/day); Cholesterol (mg/day); Self-Reported Weight (Maximum, Minimum, Weight at Age 25 Yr) In Pounds

|  | Nutrient intake total |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | White males $(n=1783)$ | Black males $(n=205)$ | White females ( $n=1930$ ) | Black females $(n=246)$ |
| Ages 46-65 |  |  |  |  |
| Quetelet inder | 2.62 (0.01) | 2.65 (0.04) | 2.61 (0.02) $\ddagger$ | 2.95 (0.06) |
| Calories | 2238 (28) $\ddagger$ | 1974 (51) | 1426 (21) | 1332 (49) |
| Protein | 89 (1.3) $\ddagger$ | 78 (2.8) | 57 (1) | 53 (2) |
| Fat | 93 (1.2) $\ddagger$ | 80 (3.1) | 59 (1) | 54 (2.6) |
| Carbohydrate | 233 (4) $\ddagger$ | 202 (8) | 160 (2.4) | 155 (5.5) |
| Saturated fat | 34 (0.5) $\ddagger$ | 29 (1.2) | 21 (0.4) | 19 (1) |
| Oleic acid | 35 (0.5) | 31 (1.3) | 22 (0.4) | 20 (1.1) |
| Linoleic acid | 12 (0.3) $\ddagger$ | 10 (0.4) | 8 (0.2) | 8 (0.4) |
| Cholesterol | 436 (11) | 472 (26) | 273 (8) | 291 (20) |
| L/O ratio | 0.37 (0.1)* | 0.34 (0.1) | 0.41 (0.01) | 0.46 (0.04) |
| Wt max | 194 (0.9) | 193 (3.3) | 158 (1.1) | 177 (2.6) |
| Wt min | 147 (0.6) | 149 (2.2) | 115 (0.5) | 126 (1.6) |
| Wt at 25 yr | 161 (0.8) | 162 (2.6) | 125 (0.6) | 135 (1.5) |
|  | White males $(n=908)$ | Black males $(n=110)$ | White females ( $n=1087$ ) | Black females $(n=128)$ |
| Ages $>65$ |  |  |  |  |
| Quetelet index | 2.55 (0.01) | 2.47 (0.05) | 2.64 (0.02) $\ddagger$ | 2.86 (.07) |
| Calories | 1832 (31) $\ddagger$ | 1590 (76) | 1297 (18) $\ddagger$ | 1182 (35) |
| Protein | 74 (1) $\ddagger$ | 63 (35) | 51 (0.8) | 49 (1.5) |
| Fat | 76 (1.5) $\dagger$ | 66 (3.5) | 50 (1.1) | 45 (2.4) |
| Carbohydrate | 204 (3.8)+ | 169 (12) | 159 (2.5) | 145 (6.7) |
| Saturated fat | 27 (0.6) | 23 (1.4) | 17 (0.4)* | 15 (0.7) |
| Oleic acid | 29 (0.7) | 25 (1.3) | 18 (0.4) | 17 (1.0) |
| Linoleic acid | 10 (0.2) | 9 (0.7) | 7.7 (0.3) | 7 (0.6) |
| Cholesterol | 382 (12) | 420 (31) | 238 (7) | 224 (21) |
| L/O ratio | 0.38 (0.01) | 0.35 (0.02) | 0.43 (0.01) | 0.45 (0.02) |
| Wt max | 186 (1.1) | 185 (2.9) | 159 (1.2) | 173 (3.4) |
| Wt min | 142 (0.9) | 143 (1.8) | 115 (0.7) | 122 (2.4) |
| Wt at 25 yr | 153 (0.9) | 153 (2.3) | 124 (0.6) | 132 (2.2) |

L 10 ratio $=$ Linoleic acid/oleic acid ratio; $\mathbf{W t}=$ weight; max $=$ maximum; min $=$ minimum.

* $p<0.02 ; \dagger p<0.01 ; \ddagger p<0.005$ ( $p$ values adjusted for multiple comparisons).

From: "Determinants of high-density lipoprotein cholesterol in blacks and whites: The second National Health and Nutrition Examination Survey", Gartside, P.S. et al. [Table III]. American Heart Journal 108(3;2):646, 1984

TABLE 28

Percent Decrease in Age-Adjusted(1) Rates For Observed(2) and Expected(3) Coronary Heart Disease Mortality Among Persons 35-74 Years of Age, According To Race and Sex: United States

Coronary heart disease mortality
Race and sex
Observed Expected

White

| Men. | 17 | 7 |
| :---: | :---: | :---: |
| Women. . . . . . . . . . . . . . . . . . . | 18 | 8 |
| Black |  |  |
| Men. . . . . . . . . . . . . . . . . . . . . . | 16 | 13 |
| Women.......................... | 24 | 16 |

${ }^{1}$ Age adjusted by direct method to the $1976-80$ National Health and Nutrition Examination Survey population.
${ }^{2}$ Percent decrease between 1973 and 1977-78.
3Estimated from risk factors measured in the 1971-75 and 1976-80 National Health and Nutrition Examination Surveys.

NOTE: Codes for coronary heart disease are 410-413 based on the Eighth Revision International Classification of Diseases, Adapted for Use in the United States.

SOURCES: National Center for Health Statistics: Data from the National Health and Nutrition Examination Survey and the National Vital Statistics System.

From: "Changes in Heart Disease Risk Factors," Rowland, M. et al. [Table D]. In: Health and Prevention Profile United States: 1983, National Center for Health Statistics/PHS/DHHS. U.S. G.P.O. pub., Washington, DC 20402, page 30

TABLE 29

Ratio of Non-White to White Median Income, United States, 1945-1977

|  |  |  | Nonwhite |  |
| :---: | :---: | :---: | :---: | :---: |
| Year | Nonwhite <br> families | Black <br> families | Males | Females |
| 1945 | 0.56 |  | n.a. | n.a. |
| 1946 | 0.59 |  | 0.61 | n.a. |
| 1947 | 0.51 |  | 0.54 | n.a. |
| 1948 | 0.53 |  | 0.54 | 0.49 |
| 1949 | 0.51 |  | 0.49 | 0.51 |
| 1950 | 0.54 |  | 0.54 | 0.49 |
| 1951 | 0.53 |  | 0.55 | 0.46 |
| 1952 | 0.57 |  | 0.55 | n.a. |
| 1953 | 0.56 |  | 0.55 | 0.59 |
| 1954 | 0.56 |  | 0.50 | 0.55 |
| 1955 | 0.55 |  | 0.53 | 0.54 |
| 1956 | 0.53 |  | 0.52 | 0.58 |
| 1957 | 0.54 |  | 0.53 | 0.58 |
| 1958 | 0.51 |  | 0.50 | 0.59 |
| 1959 | 0.52 |  | 0.47 | 0.62 |
| 1960 | 0.55 |  | 0.52 | 0.70 |
| 1961 | 0.53 |  | 0.49 | 0.67 |
| 1962 | 0.53 |  | 0.52 | 0.67 |
| 1963 | 0.53 |  | 0.57 | 0.70 |
| 1964 | 0.56 | 0.54 | 0.54 | 0.73 |
| 1965 | 0.55 | 0.54 | 0.55 | 0.76 |
| 1966 | 0.60 | 0.58 | 0.55 |  |
| 1967 | 0.62 | 0.59 | 0.59 | 0.78 |
| 1968 | 0.63 | 0.60 | 0.61 | 0.79 |
| 1969 | 0.63 | 0.61 | 0.59 | 0.85 |
| 1970 | 0.64 | 0.61 | 0.60 | 0.92 |
| 1971 | 0.63 | 0.60 | 0.61 | 0.90 |
| 1972 | 0.62 | 0.59 | 0.62 | 0.95 |
| 1973 | 0.60 | 0.58 | 0.63 | 0.93 |
| 1974 | 0.64 | 0.60 | 0.63 | 0.92 |
| 1975 | 0.65 | 0.61 | 0.63 | 0.92 |
| 1976 | 0.63 | 0.59 | 0.63 | 0.95 |
| 1977 | 0.61 | 0.57 | 0.61 | 0.88 |

n.a. $=$ Not available.

[^10]
## TABLE 30

Distribution of Family Income in Black and White Heads of Houseiold, 1978(*)

| Family income | White |  | Black |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Male head (\%) | Female head (\%) | Male <br> head <br> (\%) | Female head (\%) |
| Less than \$5,000 | 4.3 | 22.4 | 8.1 | 42.6 |
| \$5,000-\$9,999 | 13.1 | 28.0 | 18.2 | 31.7 |
| \$10,000-\$14,999 | 15.9 | 21.3 | 19.9 | 13.7 |
| \$15,000-\$19,999 | 17.9 | 13.1 | 19.2 | 6.6 |
| \$20,000-\$24,999 | 16.3 | 7.8 | 13.2 | 2.9 |
| \$25,000-\$49,999 | 28.1 | 6.9 | 20.5 | 2.5 |
| \$50,000 and over | 4.4 | 0.4 | 1.0 | $\dagger$ |
| Total $\ddagger$ | $\overline{100.0}$ | $\overline{00.0}$ | $1 \overline{00.0}$ | $\overline{100.0}$ |
| Number (thousands) | 43,636 | 5,918 | 3,244 | 2,390 |

*From money income of families and persons in the United States: 1978 current population reports, p. 60, No. 123. Washington, DC, 1980, U.S. Bureau of the Census, pp. 107-109.
$\dagger$ Less than $0.05 \%$.
$\ddagger$ May not add to 100 because of rounding.

From: "Socioeconomic influences on coronary heart disease in black populations," James, S.A. [Table I]. American Heart Journal 108(3;2):670, 1984

TABLE 31
Prevalence of Hypertension, Mean Per Capita Income(a), and Mean Age, Percent of Ideal Body Weight and Number of People Sharing the Income By Race, Sex, and Blood Pressure Level for Adults 18 Years or Older In Georgia, 1981

| Race/Sex/BP Level Prevalence | Mean Per <br> Capita <br> Income | Mean Age, <br> Weophtb, No. of <br> Pharing <br> the Income |
| :---: | :---: | :---: |

## White Men

| Normotensive $^{\text {C }}$ | 77.9 | $\$ 8221$ | $(39,107,3.1)$ | 1051 |
| :--- | ---: | ---: | :--- | ---: |
| Hypertensives $^{\text {d }}$ | 22.1 | $\$ 7865$ | $(49,117,3.0)$ | 342 |
| Controlled $^{\mathrm{e}}$ | 7.1 | $\$ 8254$ | $(56,115,2.7)$ | 94 |
| Mild $^{£}$ | 13.6 | $\$ 8012$ | $(45,117,3.1)$ | 216 |
| Moderate-Severeg | 1.4 | $\$ 4438$ | $(49,127,2.9)$ | 32 |

## White Women

Normotensive ${ }^{\mathrm{C}}$
Hypertensives
Controlled $^{\mathrm{d}}$
Mild $^{\mathrm{f}}$
Moderate-Severeg

| 81.3 | $\$ 7268$ |
| ---: | ---: |
| 17.7 | $\$ 6868$ |
| 1.0 | $\$ 7675$ |
| 6.8 | $\$ 5619$ |
| 1.0 | $\$ 6311$ |


| $(40,104,3.0)$ | 1224 |
| ---: | ---: | ---: |
| $(59,119,2.4)$ | 313 |
| $(64,116,2.1)$ | 175 |
| $(51,119,2.7)$ | 124 |
| $(50,151,3.4)$ | 14 |

## Black Men

Normotensive ${ }^{c}$
Hypertensives
Controlled $^{\mathrm{d}}$
Mild $^{\mathrm{f}}$
Moderate-Severeg

| 69.8 | $\$ 5128$ |
| ---: | ---: |
| 30.2 | $\$ 4279$ |
| 8.6 | $\$ 4670$ |
| 17.4 | $\$ 4601$ |
| 4.3 | $\$ 2196$ |


| $(34,103,3.4)$ | 430 |
| :--- | ---: |
| $(50,112,3.2)$ | 221 |
| $(59,111,2.8)$ | 62 |
| $(46,110,3.0$ | 129 |
| $(51,119,5.1)$ | 30 |

## Black Women

| Normotensive $^{\mathrm{c}}$ | 67.4 | $\$ 4048$ | $(34,112,3.3)$ | 563 |
| :--- | ---: | ---: | ---: | ---: |
| Hypertensives $^{\text {d }}$ | 32.6 | $\$ 3550$ | $(55,132,3.0)$ | 279 |
| $\quad$ Controlled $^{\mathrm{e}}$ | 18.2 | $\$ 3453$ | $(60,131,2.5)$ | 153 |
| Mild $^{\mathrm{f}}$ | 10.5 | $\$ 4448$ | $(48,130,3.4)$ | 100 |
| Moderate-Severeg | 3.9 | $\$ 1598$ | $(49,145,4.5)$ | 26 |

[^11]From: "Financial Cost as an Obstacle to Hypertension Therapy," Shulman NB, Martinez B, Brogan DR, Carr AA, Miles CG (submitted for publication)

## TABLE 32

Hypertension Detection and Follow-Up Program: Five-Year Mortality By Race and Education - Referred-Care Males Ages 40 to 69 Years, Entry DBP 90 to 104 mm Hg

| Race/education | (Deaths) N |  | 48 Mortality |  |
| :---: | :---: | :---: | :---: | :---: |
|  |  |  | Crude | Age-adjusted ${ }^{\text {a }}$ |
| WM $>$ HS | (19) | 460 | 4.1 | 4.5 |
| $W M=H S$ | (28) | 417 | 6.7 | 7.3 |
| WM < HS | (52) | 355 | 14.6 | 12.1 |
| BM < HS | (68) | 382 | 17.8 | 18.9 |

"Age-adjusted by direct method. Standard population is baseline age decade distribution of al white men aged 40 to 69 years with entry DBP 90 to 104 mmHg .

[^12]
## TABLE 33

Mean Percent of Per Capita Income Spent on Antihypertensive Drugs and Mean Reported Annual Cost of Antihypertensive Medication By Race, Sex, and Blood Pressure Level of Hypertensive Adults on Medication In Georgia, 1981

| Race/Sex/BP Level Mean | Percent Income | of Per Capita Spent on Drugs | Mean Reported Annual Cost | Sample Size <br> ( n ) |
| :---: | :---: | :---: | :---: | :---: |
| WHITE MEN | 2.27 |  | \$166 | (128) |
| Controlled ${ }^{\text {b }}$ |  | 2.2\% | \$179 | (85) |
| Mild ${ }^{\text {c }}$ |  | 1.6\% | \$ 97 | (35) |
| Moderate to Severe ${ }^{\text {d }}$ |  | 8.78 | \$338 | (8) |
| WHITE WOAEN | 2.18 |  | \$149 | (205) |
| Controlled ${ }^{\text {b }}$ |  | $2.0 \%$ | \$147 | (156) |
| Mild ${ }^{\text {c }}$ |  | 2.8\% | \$154 | (45) |
| Moderate to Severe ${ }^{\text {d }}$ |  | 3.48 | \$208 | (4) |
| BLACK MLEN | $2.6 \%$ |  | \$123 | (85) |
| Controlled ${ }^{\text {b }}$ |  | $2.6 \%$ | \$130 | (54) |
| Mild ${ }^{\text {c }}$ |  | 1.9\% | \$ 94 | (24) |
| Moderate to Severe ${ }^{\text {d }}$ |  | $5.0 \%$ | \$155 | (7) |
| 8LACK WONIEN | 3.98 |  | \$140 | (189) |
| Controlled ${ }^{\text {b }}$ |  | 3.18 | \$106 | (138) |
| Mild ${ }^{\text {c }}$ |  | 4.4\% | \$231 | (39) |
| Moderate to Severe ${ }^{\text {d }}$ |  | 19.6\% ${ }^{\text {e }}$ | \$235 | (12) |

a. Percent of Per Caples Income Spent on Drugs. Annual Prescription Cost $\times 100 \%$

Per Capita Income
b. Controlled $=$ DBP less than 90 me Hg and on antiliypertensive medication.
c. Mild - DAP 90 to $104 \mathrm{~mm} \mathrm{Hg}_{\mathrm{g}}$ and on antihypertensive medicacion.
d. Hoderate to Severe - DBP at least 105 Hg and on antihypertensiveg medication.
e. One of the twelve black women in the moderate to severe group reported epending $54 \%$ of her per rapita income on antihypertensive medication. If ahe is conaidered an outlier, the remalning eleven women yleld a mean percent of 6.49 rather than $19.6 \%$ and the an for the 188 black women is $3.5 \%$ rather than 3.9\%.

[^13]TABLE 34
Selected Demographic Groups

Response

| Total | $\frac{\text { Less than }}{\text { high school }}$ |  |  |
| :--- | :--- | :--- | :--- |
| $\frac{\text { sample, }}{\text { percent }}$ | $\frac{\text { Blacation, }}{\text { edercent }}$ | $\frac{\text { Age }}{\text { percent }}$ | $\frac{\text { Age }}{\text { percent }}$ |

Awareness

| Heard hypertension is diet-related | 81 | 71 | 79 | 84 | 72 |
| :--- | :--- | :--- | :--- | :--- | :--- |
| Name salt/sodium as factor | 54 | 42 | 52 | 56 | 43 |
| Name alcohol as factor | 26 | 21 | 11 | 27 | 22 |
| Name fats as factor | 17 | 15 | 15 | 16 | 15 |
| Name cholesterol as factor | 17 | 8 | 10 | 20 | 10 |
| Heard other CVD and diet-related | 58 | 39 | 48 | 55 | 54 |
| Name cholesterol as factor | 26 | 11 | 14 | 29 | 22 |
| Name fats as factor | 18 | 11 | 13 | 18 | 17 |
| Name alcohol as factor | 13 | 10 | 14 | 12 | 14 |
| Name salt/sodium as factor | 11 | 10 | 10 | 11 | 10 |
| Heard salt/sodium may be harmful | 73 | 62 | 70 | 72 | 65 |
| $\quad$ Name hypertension | 51 | 44 | 57 | 48 | 41 |
| Heard cholesteral or saturated |  |  |  |  |  |
| fats may be harmful | 63 | 47 | 51 | 64 | 53 |
| Name heart/coronary problems | 42 | 27 | 22 | 47 | 27 |
| Name atherosclerotic disease | 26 | 12 | 16 | 33 | 15 |
|  |  |  |  |  |  |
| Concern |  |  |  | 63 | 48 |
| Concerned with salt/sodium | 61 | 55 | 56 | 27 |  |
| On medically prescribed diet | 12 | 16 | 15 | 50 | 59 |
| Concerned with fat | 60 | 49 | 65 |  |  |
| On medically prescribed diet | 13 | 15 | 21 | 6 | 20 |
| Concerned with cholesterol | 65 | 57 | 67 | 65 | 67 |
| On medically prescribed diet | 12 | 17 | 17 | 4 | 27 |

From: Cardiovascular Disease and Diet, Heimbach, J.T. [Table 14]. Public Health Reports, January 1985
TABLE 35
Number and Percent Distribution of Office Visits for Diseases of the Circulatory System By Age, Sex, and Race of Pat ient, According to
Selected Principal Diagnoses: United States, 1975-76

|  |  |  | Age |  |  |  |  |  | Sex |  | Race |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Principal diagnosis and ICDA code ${ }^{1}$ | Number of visits in thousands | Total | Under 35 years | $\begin{aligned} & 35-44 \\ & \text { years } \end{aligned}$ | $\begin{aligned} & 45-54 \\ & \text { years } \end{aligned}$ | $55-64$ <br> years | $\begin{aligned} & 65-74 \\ & \text { years } \end{aligned}$ | 75 years and over | Male | Female | White | Female |
|  |  | Percent distribution |  |  |  |  |  |  |  |  |  |  |
| Essential benign <br> hypertension ........ 401 | 46,128 | 100.0 | 5.9 | 8.2 | 19.9 | 30.2 | 25.2 | 10.6 | 63.5 | 36.5 | 87.6 | 12.4 |
| Acute ischemic <br> heart disease.... 410-411 | 2,319 | 100.0 | *2. 1 | *6.9 | 21.2 | 26.4 | 29.9 | *13.5 | 42.9 | 57.1 | 94.0 | 6.0 |
| Chronic ischemic <br> heart disease ....... 412 | 26,020 | 100.0 | * 1.2 | 3.6 | 14.3 | 23.2 | 31.3 | 26.4 | 47.8 | 52.2 | 92.2 | 7.8 |
| Angina pectoris ....... 413 | 2,975 | 100.0 | *2.1 | *3.2 | 28.9 | 25.0 | 28.2 | *12.6 | 47.0 | 53.0 | 92.5 | 7.5 |
| Symptomatic heart <br> disease ............. 427 | 7,052 | 100.0 | 9.0 | *6.7 | 11.0 | 18.6 | 31.2 | 23.6 | 51.1 | 48.9 | 91.5 | 8.5 |
| Cerebrovascular <br> disease ......... 430-438 | 4,505 | 100.0 | *2.0 | * 1.0 | * 10.6 | 18.1 | 29.3 | 38.9 | 49.6 | 50.4 | 94.0 | 6.0 |
| Arteriosclerosis ...... 440 | 2,019 | 100.0 | *0.5 | *4.5 | *2.5 | *12.1 | 29.8 | 50.6 | 63.4 | 36.6 | 94.7 | 5.3 |
| Phlebitis and thrombophlebitis .... 451 | 2,930 | 100.0 | *14.8 | *12.2 | 23.3 | 20.1 | 21.6 | *8.0 | 68.7 | 31.3 | 93.5 | 6.5 |
| Varicose veins of lower extremities.... 454 | 2,428 | 100.0 | *14.2 | *13.6 | 21.9 | 25.7 | *15.2 | *9.3 | 81.5 | 18.5 | 93.2 | 6.8 |
| Hemorrhoids ........... 455 | 3,686 | 100.0 | 29.5 | 19.1 | 23.5 | 14.2 | *10.6 | *3.1 | 44.4 | 55.6 | 91.2 | 8.8 |

liagnostic groupings and code inclusions are based on the Eighth Revision International Classification of Diseases, Adapted for Use in the United States, 1965.
From: "Office Visits for Diseases of the Circulatory System, the National Ambulatory Medical Care Survey, United January 1979
TABLE 36


| State | Sex | Year | Age$(y r)$ | Ratios of black to white percentages in hypertensive patients |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  |  | \% | Unaware $\dagger$ | \% Untreated but aware | \% Uncontrolled although treated | \% "Definite" $\ddagger$ but uncontrolled |
| South | M | 1979 | 18+ |  | 0.91 | 0.81 | 1.12 | 1.08 |
| Carolina | M | 1982 | 18+ |  | 1.14 | 0.99 | 1.18 | 1.53 |
|  | F | 1979 | 18+ |  | 0.85 | 1.13 | 1.12 | 1.11 |
|  | F | 1982 | 18+ |  | 0.76 | 1.02 | 1.12 | 1.43 |
| California | M | 1979 | 50+ |  | 0.72 | 0.85 | 0.97 | - |
|  | F | 1979 | 50+ |  | 0.60 | 0.47 | 1.12 | -- |
| Georg ia | M | 1981 | $50+$ |  | 0.75 | 1.04 | 1.20 | 1.78 |
|  | F | 1981 | 50+ |  | 0.64 | 0.87 | 1.03 | 1.48 |
| Connecticut | M | 1979 | $50+$ |  | 0.76 | 0.49 | 0.98 | 1.14 |
|  | M | 1982 | $50+$ |  | 0.90 | 1.29 | 0.71 | 0.71 |
|  | F | 1979 | 50+ |  | 0.88 | 1.10 | 1.06 | 0.93 |
|  | F | 1982 | 50+ |  | 0.23 | 0.85 | 0.99 | 0.57 |

*Unpublished data from Daniel H. Freeman, Ph.D., Connecticut High Blood Pressure Program. $\dagger$ Defined as $140 / 90 \mathrm{~mm} \mathrm{Hg}$ or on medication. †Defined as $160 / 95 \mathrm{~mm} \mathrm{Hg}$.

FIGURE 1

Death By Cause and Percentage of Total Deaths

1968


1978*


- Basec on Provismon Date
- Coded in 1968 as Bronchites and Emphivema

Sources: National Center for Health Statistics National Heart, Lung, and Blood Institute Percentages do not add to $100 \%$ because of rounding.

From: "The Decline in Cardiovascular Disease Mortality", Levy, R.I. [Figure 1]. Annual Review of Public Health 2:50, 1981

FIGURE 2

Numbers of Deaths Among United States' Blacks and Other Minorities In 1977 (Blacks Comprise $87 \%$ of This Group)


Total Deaths 235,497

From: "Coronary Heart Disease in Black Populations: Mortality and Morbidity", Gillum, R.F. [Figure 1]. American Heart Journal 104(4;1):840, 1982

## FIGURE 3

Age-Adjusted CHD Mortality Rates Per 100,000 Population For U.S. NonWhites Aged 34 to 74 Years, From 1940 to 1978


From: "Coronary heart disease mortality in United States blacks, 1940 1978: Trends and unanswered questions", Gillum, R.F. and Liu, K.C. [Figure 1]. American Heart Journal 108(3;2):729, 1984

FIGURE 4

Age-Adjusted CHD Incidence Rates in Black Males (BM), White Males (WM), High Socioeconomic Status Black Males (BMH), Black Women (BW), and White Women (WW)


From: "Incidence of coronary heart disease in blacks in Charleston, South Carolina", Keil, J.E. et al. [Figure 3]. American Heart Journal 108(3;2):781, 1984

FIGURE 5

Age-Adjusted Sudden CHD Death Rates Per Thousand By Race and Sex, Nashville, 1967-1968


From: "Coronary heart disease mortality in United States blacks, 19401978: Trends and unanswered questions", Gillum, R.F. and Liu, K.C. [Figure 4]. American Heart Journal 108(3;2):731, 1984

FIGURE 6

Average End-Stage Renal Disease Incidence Rates, By Primary Etiology, Race, and Sex (1973-1979 Average)


[^14]
## FIGURE 7

Five-Year Age-Adjusted CHD Mortality Rate (Per 1,000) By Diastolic Blood Pressure Level By Race

No. Black Men: 3,602
No. White Men: 67,418
3,734
65,242
3,955
65,051
7,491

From: "Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years", Neaton, J.D. et al. [Figure 2]. American Heart Journal 108(3;2):763, 1984

## FIGURE 8

Mean Serun Cholesterol Levels in Quintile Strata of Body Mass Index For Adults 18-74 Years of Age By Race and Sex: United States, 1971-74. NHANES I


From: "Dietary Intake and Cardiovascular Risk Factors, Part II. Serum Urate, Serum Cholesterol, and Correlates; United States 1971-1975." National Center for Health Statistics, WR Harlan, AL Hill, RP Schmouder et al. Vital and Health Statistics, Series II, No. 227. DHHS Pub. No. (PHS) 83-1677, March 1983

## FIGURE 9

Five-Year Age-Adjusted CHD Mortality Rate (Per 1,000) By Serum Cholesterol Level By Race


From: "Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years", Neaton, J.D. et al. [Figure 5]. American Heart Journal 108(3;2):765, 1984

FIGURE 10

Five-Year Age-Adjusted CHD Mortality Rate (Per 1,000) By Number Of Cigarettes Reported Smoked By Race


From: "Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years", Neaton, J.D. et al. [Figure t]. American Heart Journal 108(3;2):767, 1984

## FIGURE 11

## CHD Mortality Ratios for Black and White Males and Females By Amount of Cigarette Smoking Per Day



Fig. 1. CHD mortality ratios for males by amount of cigarette smoking per day, by race.


Fig. 2. CHD mortality ratios for females by amount of cigarette smoking per day, by race.


Fig. 3. CHD mortality ratios for males by amount of cigarette smoking per day in subjects with no history of serious disease, by race.


Fig. 4. CHD mortality ratios for females by amount of cigarette smoking per day in subjects with no history of serious disease, by race.

FIGURE 12

Quartiles in the Distribution of Body Mass Index of White and Black Males and Females, 18-74 Years, By Age: United States, 1971-74


From: "Dietary Intake and Cardiovascular Risk Factors, Part I. Blood Pressure Correlates; United States, 1971-1975." Fig. 1. National Center for Health Statistics, WR Harlan, AL Hill, RP Schmouder et al. Vital and Health Statistics, Series II, No. 226. DHHS Pub. No. (PHS) 83-1676, Feb 1983

FIGURE 13

Five-Year Age-Adjusted Cerebrovascular Disease Mortality Rate (Per 1,000) By Diastolic Blood Pressure Level By Race


No. Black Men: 3,602
No. White Men: 67,418
65,242
61,351
65,051
66,322

FIGURE 14

Five-Year Age-Adjusted Cerebrovascular Disease Mortality Rate (Per 1,000) By Serum Cholesterol Leve1 By Race


Serum Cholesterol Quintile ( $\mathrm{mg} / \mathrm{dl}$ )

No. Black Men: 5,947
No. White Men: 62,614

| 4,835 | 4,214 | 4,117 | 4,377 |
| ---: | ---: | ---: | ---: |
| 65,222 | 64,277 | 66,011 | 67,260 |

From: "Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years", Neaton, J.D. et al. [Figure 6]. American Heart Journal 108(3;2):765, 1984

## FIGURE 15

Prevalence of Hypertension at First Screen By Years of Education. DBP > or $=95 \mathrm{~mm}$ Hg and/or Reporting Current Use of Antihypertensive Medication


From: "Race, Education, and Prevalence of Hypertension", the Hypertension Detection and Follow-Up Program Cooperative Group. Figure 1. American Journal of Epidemiology 106(5):351-361, 1977

## FIGURE 16

Stroke Mortality For Black Males in North Carolina By County Index Of Social Disorganization, 1956-1964.


From: "The Contribution of the Social Environment To Host Resistance", Cassel, J. [Figure 3]. American Journal of Epidemiology 104(2): 115, 1976

FIGURE 17

Hypertension-Related Deaths Per 10,000 By Socioecologic Stress Levels In Non-White Males Aged 45-54 in North Carolina, 1960


Figure adapted by permission from James and Kleinbaum American Journal of Public Health 66(4):354-358, 1976

[^15]
## FIGURE 18

1982 Percent of Total Persons Below the Poverty Level By Race


Source: U.S. Bureau of the Census

FIGURE 19

Average Annual Rate of Office Visits For Selected Diseases of the Circulatory System, By Race of Patient: United States, 1975-76


Principal Diagnosis and ICDA Code

[^16]
## Hispanic Americans

Tables 40-42

TABLE 40

Five Leading Causes of Death, By Race-Ethnicity, Los Angeles, 1981

| Rank | White |  | Black | Asian-Americans |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Non-Spanish- | Spanish- |  |  |  |  |
|  | surnamed* | surnamed* |  | Japanese | Chinese | Korean |
| 1 | Diseases of heart $(42)^{+}$ | Diseases of heart (27) | Diseases of heart (31) | Diseases of heart (32) | Diseases of heart (32) | Malignant neoplasms (25) |
| 2 | Malignant neoplasms (22) | Malignant neoplasms (18) | Malignant neoplasms (21) | Malignant neoplasms (22) | Malignant neoplasms (27) | Diseases of heart (16) |
| 3 | Cerebrovascular diseases (9) | Accidents <br> (9) | Cerebrovascular diseases (8) | ```Cerebrovascu- lar diseases (15)``` | Cerebrovascu- <br> lar diseases <br> (11) | Cerebrovascular diseases (10) |
| 4 | Accidents (4) | Cerebrovascular diseases (6) | Accidents <br> (6) | Accidents <br> (5) | Accidents (5) | Accidents <br> (8) |
| 5 | Pneumonia and influenza (3) | Chronic liver disease and cirrhosis (5) | Chronic liver disease and cirrhosis (3) | Pneumonia and influenza (4) | Suicide <br> (4) | Suicide <br> (4) |

*As identified on the death certificate
+Percentage of total deaths in the specified race-ethnic group.

[^17]
## TABLE 41

Age-Adjusted Mortality Rates Among Males By Race-Ethnicity,
Los Angeles County, 1979-81

| Causes of cesth (ICD Code Nunber) | Age-adjustad cortallty rates per 100,000 anles population |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Los County | nito | Breck | Mispenlet | Astan-Amoricana |  |  |
|  |  |  |  |  | depanese | Cinese | Roram |
| All csuses | ${ }_{(03,441)^{*}}^{845.5}$ | $\begin{gathered} 1103.5 \\ (78,516) \end{gathered}$ | $\begin{gathered} 1327.4 \\ (12,324) \end{gathered}$ | $\begin{array}{r} 995.7 \\ (5,684) \end{array}$ | $\begin{aligned} & 624.1 \\ & 1876) \end{aligned}$ | $\begin{aligned} & 49.0 \\ & \text { (434) } \end{aligned}$ | $\begin{aligned} & 532.8 \\ & \text { (203) } \end{aligned}$ |
| Mojor cardlorescular diseaseal (390-44) | $\begin{array}{r} 405.7 \\ (42,872) \end{array}$ | $\begin{gathered} 536.6 \\ (37,308) \end{gathered}$ | $\begin{array}{r} 550.2 \\ (4,444) \end{array}$ | $\left(\begin{array}{c} 441.9 \\ (2.204) \end{array}\right.$ | $\begin{aligned} & 323.2 \\ & (430) \end{aligned}$ | $\begin{aligned} & 228.1 \\ & (1144) \end{aligned}$ | $\begin{aligned} & 169.0 \\ & (49) \end{aligned}$ |
| Disassos of hact <br> (300-396, 402. 404-429) | $\begin{gathered} 322.4 \\ (34,765) \end{gathered}$ | $\begin{gathered} 432.6 \\ (30,392) \end{gathered}$ | $(3,552)$ | $\begin{array}{r} 357.1 \\ (1,795) \end{array}$ | $\begin{aligned} & 220.4 \\ & 6011 \end{aligned}$ | $\begin{aligned} & 178.9 \\ & (140) \end{aligned}$ | $\begin{array}{r} 117.2 \\ (30) \end{array}$ |
| Ischemle heart disease (IMD) (410-814) | $\begin{gathered} 201.8 \\ (21,120) \end{gathered}$ | $\begin{array}{r} 274.0 \\ (18.951) \end{array}$ | $\left(\begin{array}{c} 223.9 \\ (1,704) \end{array}\right.$ | $\begin{aligned} & 200.4 \\ & \text { (903) } \end{aligned}$ | $\begin{aligned} & 147.7 \\ & 1541 \end{aligned}$ | $\begin{aligned} & 77.9 \\ & \text { (77) } \end{aligned}$ | $\begin{aligned} & 78.1 \\ & \text { (17) } \end{aligned}$ |
| mpocardisl inferction and esute IHD (410, 411) | $\begin{array}{r} 95.7 \\ (10,739) \end{array}$ | $\begin{gathered} 139.7 \\ (9,658) \end{gathered}$ | $\begin{aligned} & 105.9 \\ & \text { (840) } \end{aligned}$ | $\begin{array}{r} 98.2 \\ (497) \end{array}$ | $\begin{array}{r} 74.5 \\ (100) \end{array}$ | $\begin{aligned} & 48.3 \\ & \text { (41) } \end{aligned}$ | 24.4 <br> (1) |
| Oranle INo (412-414) | $\begin{gathered} 106.1 \\ (10,309) \end{gathered}$ | $\begin{gathered} 13, .3 \\ (9,293) \end{gathered}$ | $\begin{aligned} & 117.0 \\ & (864) \end{aligned}$ | $\begin{aligned} & 102.2 \\ & 1485) \end{aligned}$ | $\begin{aligned} & 73.5 \\ & \text { (34) } \end{aligned}$ | $\begin{aligned} & 49.6 \\ & \text { (3) } \end{aligned}$ | $\begin{aligned} & 53.8 \\ & (10) \end{aligned}$ |
| Mpertensive slisessel (401-404) | $(20,171)$ | $\begin{gathered} 22.0 \\ (1.610) \end{gathered}$ | (4799) | (20.4) | 16.4 | (16) | 2.8 |
| Cerchrovaseular (Isasses (stroken) (330-430) | $(5,971)$ | $\begin{gathered} 73.4 \\ (5,0 i 5) \end{gathered}$ | $\begin{aligned} & 84.6 \\ & (713) \end{aligned}$ | (309) | $(107)$ | 33.3 (32) | $\begin{aligned} & 33.7 \\ & (17) \end{aligned}$ |
| Mallonant neoplams leancer) | $\begin{gathered} 157.8 \\ (19,165) \end{gathered}$ | $\begin{gathered} 210.5 \\ (16,155) \end{gathered}$ | $\begin{array}{r} 285.9 \\ (2,423) \end{array}$ | $\begin{aligned} & 182.9 \\ & (979) \end{aligned}$ | $\begin{aligned} & 141.9 \\ & \text { (214) } \end{aligned}$ | $\begin{aligned} & 127.2 \\ & (115) \end{aligned}$ | $\begin{aligned} & 157.8 \\ & (53) \end{aligned}$ |
| Preumonio and luftcenze (400-477) | $(2,285)$ | $(1,946)$ | $\begin{gathered} 32.0 \\ (234) \end{gathered}$ | $\begin{gathered} 30.1 \\ (169) \end{gathered}$ | $\begin{aligned} & 21.7 \\ & \text { (28) } \end{aligned}$ | $\begin{aligned} & 11.4 \\ & \text { (1) } \end{aligned}$ | 17.6 <br> (3) |
| Cironle Ilver flagese end elrrtiosia (571) | $(24.3$ | $(2,484)$ | $\begin{gathered} 37.0 \\ (411) \end{gathered}$ | $\begin{aligned} & 99.3 \\ & \text { (356) } \end{aligned}$ | (i3) | $\begin{aligned} & 9.4 \\ & (3) \end{aligned}$ | $7.9$ <br> (5) |
| Aceldents and adverse offects (E800-E949) | $(5,183)$ | $(5,0701$ | $\begin{array}{r} 73.6 \\ (354) \end{array}$ | $\begin{gathered} 61.6 \\ (471) \end{gathered}$ | $\begin{aligned} & 20.0 \\ & (36) \end{aligned}$ | $\begin{aligned} & 20.6 \\ & 131) \end{aligned}$ | $\begin{aligned} & 52.5 \\ & (25) \end{aligned}$ |


t Census trects ls whleh 758 or core of the population ore persens of Spenish/Mispeale origle or coscent

- number of ceane in parentheses
 efthout (403) renal ilseasc.

From: "Cardiovascular Diseases in Los Angeles County, 1978-1981", Frerichs, R.R. et al. American Heart Association. Greater Los Angeles Affiliate, Inc., 1983

TABLE 42

```
Age-Adjusted Mortality Rates Among Females By Race-Ethnicity,
    Los Angeles County, 1979-81
```

| Couses of couth (100 006e Mmber) |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\begin{aligned} & \text { Los } \\ & \text { Angeles } \\ & \text { County } \end{aligned}$ | -180 | Olmek | Mispenie ${ }^{\text {P }}$ | As lon-herlcas |  |  |
|  |  |  |  |  | dupenese | Olmes | Beren |
| All ceuses | $\begin{gathered} 631.0 \\ (18,088) \end{gathered}$ | $\begin{gathered} 667.2 \\ (74,099) \end{gathered}$ | $\begin{gathered} 772.5 \\ (0.332) \end{gathered}$ | $(07.0$ | $\begin{aligned} & 382.2 \\ & \text { (690) } \end{aligned}$ | $\begin{aligned} & 380.0 \\ & (353) \end{aligned}$ | $\begin{aligned} & 309.7 \\ & \text { (150) } \end{aligned}$ |
| Mem corilonsewler ilsanent (390-44) | $\begin{gathered} 324.3 \\ 14.6811 \end{gathered}$ | $\begin{gathered} 33.7 \\ (41.455) \end{gathered}$ | $\begin{gathered} 34.4 \\ (4,371) \end{gathered}$ | $\begin{gathered} 316.7 \\ (2,345) \end{gathered}$ | $\begin{aligned} & 109.4 \\ & \text { (374) } \end{aligned}$ | 171.7 <br> (135) | 121.1 (42) |
| $\begin{aligned} & \text { Dicatcen of meat } \\ & \quad 350-398, \cos , 04-691 \end{aligned}$ | 233.25.4 | $\begin{gathered} 245,0 \\ (30,1731 \end{gathered}$ | $(278.0$ | $\begin{aligned} & 24.4 \\ & (1.742) \end{aligned}$ | $\begin{aligned} & 118.6 \\ & (284) \end{aligned}$ | $\log _{4} 4$ | $\begin{aligned} & 1.0 \\ & \text { (t) } \end{aligned}$ |
| Ischele meort dicense (IMOI ( $610-414$ ) | $\begin{gathered} 190.9 \\ 121.241 \end{gathered}$ | $\begin{gathered} 198.1 \\ (18.717) \end{gathered}$ | $\begin{gathered} 188.5 \\ (1.773) \end{gathered}$ | $\begin{aligned} & 14.6 \\ & (1,09) \end{aligned}$ | $\begin{array}{r} 2.1 \\ 11601 \end{array}$ | C4.4 | $\begin{aligned} & \text { ea.1 } \\ & \text { (i8) } \end{aligned}$ |
| Mpoeprilal lafortion end exte IND (410, 411) | $(5,4101$ | $\begin{gathered} 30.1 \\ (0,42) \end{gathered}$ | $\begin{aligned} & 70.9 \\ & \text { reos } \end{aligned}$ | es. 8 | 38.4 | 38.2 | 820 |
| Emonic ino (412-414) | $13.5$ | $(11.275)$ | 77.6 | 14.07) | 45.7 | 2.28 | $\begin{array}{r} 27.6 \\ \text { (8) } \end{array}$ |
| Mrertensive ilemsol (401-4) | $\begin{gathered} 17,2 \\ (2,3791 \end{gathered}$ | $\begin{gathered} 13.8 \\ 11,8821 \end{gathered}$ | 0988 | 19.6) | 7.3 1153 | 14.4 | 3.1 |
| Orabrovascular flaceses (stranea) (430-43) | $(10,104)$ | $\begin{gathered} 71.0 \\ (8,206) \end{gathered}$ | 4.6 61 | 578) | (120) | 238) | $\begin{aligned} & 3.4 \\ & (15) \end{aligned}$ |
| ```Mligment meoplam:s (ememp) (140-200)``` | $\begin{gathered} 144,0 \\ (10,67) \end{gathered}$ | $\begin{gathered} 154.8 \\ (16,182) \end{gathered}$ | $\begin{gathered} 18.9 \\ (1.874) \end{gathered}$ | 115.0 cals | (18.3 | 0.1 | 859 |
|  | $\begin{gathered} 17.3 \\ 4,5321 \end{gathered}$ | $(2,28.4)$ | 19.4 | 19.3 (145) | 0.0 | 11.3 | 15.3 |
|  (871) | $(1,487$ | $\begin{gathered} 13.5 \\ (1,28) \end{gathered}$ | 1380 | 17.7 (128) | 3.4 | 45 | 78 |
| aecillants and etverse offects (500-ET49) | (2,0.051 | $\begin{gathered} 23.7 \\ (2.189) \end{gathered}$ | 24.6 8541 | $\begin{aligned} & 81.0 \\ & \text { (173) } \end{aligned}$ | 13.1 | 18.8 | $\begin{aligned} & 16.0 \\ & \text { (11) } \end{aligned}$ |




- members of chathe in perentheess
 olthent (co3) renal ilsoese.

[^18]
## TABLES

Asian/Pacific Islander Americans
Tables 50-72

TABLE 50

Percent of Population Completing 4 Years or More of College By Specified Race, Age, and Sex: United States, 1980

| Race and Ase | K. 1. |  | Fenale |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Total | Percent | Fotal | Percent |
| Whitel |  |  |  |  |
| 25-34 years | 15,400, 161 | 24.5 | 15,394,841 | 21.7 |
| 35-44 years | 10,711,364 | 25.9 | 10,930,907 | 15.6 |
| 45-64 years | 18,618,917 | 18.2 | 20,292,624 | 9.6 |
| 65 years and over | 9,210,721 | 10.5 | 13,730,849 | 7.6 |
| Chinese ${ }^{2}$ |  |  |  |  |
| 25-34 years | 4,453 | 57.1 | 4,758 | 45.6 |
| 35-44 years | 2,601 | 55.0 | 2,619 | 34.4 |
| 45-64 years | 3,742 | 30.7 | 3.552 | 15.0 |
| 65 years and over | 1,391 | 18.5 | 1.450 | 6.8 |
| Jupanese ${ }^{2}$ |  |  |  |  |
| 25-34 yesrs | 3,287 | 49.3 | 3,517 | 40.4 |
| 35-44 years | 1,939 | 48.9 | 2,861 | 24.7 |
| 65-64 years | 3,878 | 23.7 | 5,827 | 8.9 |
| 65 years and over | 1,164 | 7.9 | 1,642 | 4.6 |
| Pilipino ${ }^{2}$ |  |  |  |  |
| 25-34 years | 3,374 | 33.2 | 4,832 | 46.3 |
| 35-44 years | 2,740 | 47.6 | 3,412 | 53.5 |
| 65-64 yeara | 2,015 | 31.9 | 2,911 | 27.9 |
| 65 years and over | 1,880 | 8.1 | 982 | 11.2 |

1 Compiled from published census reports.
2Data are from the 1980 Census Fublic Die Microdata A (5\%) ample, couputed Ey 8. Kan.

[^19]
## TABLE 51

|  | Chinese |  |  | Japanese |  |  | Filipino |  |  | White |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | U.S. born | Foreign born | Total | $\begin{aligned} & \text { U.S. } \\ & \text { born } \end{aligned}$ | $\begin{aligned} & \text { Foreign } \\ & \text { born } \end{aligned}$ | Total | U.S. born | Foreign born | Total |  |
| Professional | 19.9 | 18.3 | 18.7 | 15.5 | 13.7 | 15.0 | 7.4 | 18.4 | 16.3 | 12.8 |
| Executive, administrative, and managerial | 12.6 | 11.5 | 11.8 | 10.7 | 13.6 | 11.5 | 6.7 | 7.2 | 7.1 | 11.1 |
| Technical | 6.3 | 6.0 | 6.1 | 4.2 | 3.8 | 4.1 | 3.5 | 5.8 | 5.3 | 3.1 |
| Administrative support, including clerical | 23.1 | 13.0 | 15.7 | 21.9 | 13.9 | 19.7 | 21.6 | 20.8 | 20.9 | 17.3 |
| Sales | 11.9 | 8.3 | 9.2 | 10.7 | 10.2 | 10.5 | 10.0 | 5.3 | 6.3 | 10.7 |
| Precision production, craft, and repair | 6.4 | 5.1 | 5.5 | 10.7 | 6.4 | 9.5 | 11.4 | 7.3 | 8.1 | 13.4 |
| Operators, fabricators, and laborers | 7.4 | 15.5 | 13.3 | 10.2 | 13.7 | 11.1 | 16.7 | 14.2 | 14.7 | 17.1 |
| Farming, forestry, and fishing | 1.0 | 0.6 | 0.7 | 5.0 | 3.5 | 4.6 | 3.6 | 3.7 | 3.7 | 2.9 |
| Service | 11.2 | 21.1 | 18.5 | 10.6 | 20.1 | 13.2 | 18.8 | 16.8 | 17.2 | 11.3 |
| Private household occupations | 0.3 | 0.7 | 0.6 | 0.6 | 1.0 | 0.0 | 0.3 | 0.7 | 0.6 | 0.4 |
| (W) | $(6,607)$ | $(18,062)$ | $(24,669)$ | ( 16,810 ) | $(6,353)$ | $(23,163)$ | $(4,219)$ | (17,481) | ( 21,700 ) | $(84,027,375)$ |

## TABLE 52

Family Income For Three Asian American Groups By Nativity, and For the

|  | Chinese |  |  | Japanese |  |  | Filipino |  |  | White |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\overline{\text { U.S. }}$ born | $\begin{aligned} & \text { Foreign } \\ & \text { born } \end{aligned}$ | Total | $\begin{aligned} & \overline{\text { U.S. }} \\ & \text { born } \end{aligned}$ | Foreign born | Total | U.S. born | Foreign born | Total |  |
| Median family income | \$20,955 | \$21,010 | \$22,910 | \$29,373 | \$21,195 | \$27,475 | \$21,310 | \$24,010 | \$23,585 | \$28,835 |
| Percent of families with income |  |  |  |  |  | 3.1 | 5.3 | 3.3 | 3.7 | 5.6 |
| Less than \$5,000 | 3.1 | 6.6 | 5.9 | 1.9 | 6.4 | 57.0 | 38.6 | 48.1 | 46.6 | 37.8 |
| \$25,000 or more | 60.1 | 41.5 | 45.4 | 62.9 | 40.7 | 57.0 | 38.6 | 48.1 | 46.6 | 37.8 |
| (W) | $(1,981)$ | $(7,368)$ | $(9,349)$ | $(6,176)$ | $(2,209)$ | $(8,385)$ | $(1,349)$ | $(7,032)$ | $(8,381)$ | $(50,644,862)$ |

Mortality, U.S., 1979-1981: Excess Deaths From Heart Disease

| Age | $\begin{gathered} 390 \text { to } 398402404 \text { to } 429 / \text { Heart Disease } \\ \text { Race }=\text { White } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{array}{r} \text { Ratel } \\ 100,000 \\ \hline \end{array}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{array}{r} \text { Rate/ } \\ 100,000 \\ \hline \end{array}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 303 | 21.71 | 0. | 0. | * | * | 219 | 16.54 | 0. | 0. | * | * |
| 1-4 | 111 | 2.19 | 0. | 0. | * | * | 103 | 2.14 | 0. | 0. | * | * |
| 5-9 | 55 | 0.82 | 0. | 0. | * | * | 53 | 0.83 | 0. | 0. | * | * |
| 10-14 | 77 | 1.03 | 0. | 0. | * | * | 54 | 0.77 | 0. | 0. | * | * |
| 15-19 | 188 | 2.17 | 0. | 0. | * | * | 111 | 1.34 | 0. | 0. | * | * |
| 20-24 | 317 | 3.65 | 0. | 0. | * | * | 176 | 2.05 | 0. | 0. | * | * |
| 25-29 | 498 | 6.22 | 0. | 0. | * | * | 240 | 3.01 | 0. | 0. | * | * |
| 30-34 | 1021 | 13.99 | 0. | 0. | * | * | 400 | 5.45 | 0. | 0. | * | * |
| 35-39 | 2258 | 38.72 | 0. | 0. | * | * | 652 | 10.99 | 0. | 0. | * | * |
| 40-44 | 4615 | 95.16 | 0. | 0. | * | * | 1225 | 24.62 | 0. | 0. | * | * |
| 45-49 | 9218 | 198.73 | 0. | 0. | * | * | 2385 | 49.49 | 0. | 0. | * | * |
| 50-54 | 17395 | 353.70 | 0. | 0. | * | * | 4958 | 94.63 | 0. | 0. | * | * |
| 55-59 | 28276 | 582.67 | 0. | 0. | * | * | 9444 | 175.38 | 0. | 0. | * | * |
| 60-64 | 38527 | 923.22 | 0. | 0. | * | * | 15993 | 333.01 | 0. | 0. | * | * |
| 65-69 | 49198 | 1413.08 | 0. | 0. | * | * | 25260 | 583.28 | 0. | 0. | * | * |
| 70-74 | 55227 | 2163.78 | 0. | 0. | * | * | 37014 | 1044.69 | 0. | 0. | * | * |
| 75-79 | 53635 | 3249.85 | 0. | 0. | * | * | 49236 | 1851.04 | 0. | 0. | * | * |
| 80-84 | 46245 | 5008.62 | 0. | 0. | * | * | 59807 | 3394.17 | 0. | 0. | * | * |
| 85-Plus | 54353 | 8846.47 | 0. | 0. | * | * | 105794 | 7395.32 | 0. | 0. | * | * |
| All Ages | 361516 | 394.30 | 0. | 0. | * | * | 313124 | 323.86 | 0. | 0. | * | * |

Race = Black

|  | Male |  |  |  |  |  | Black Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Age | Deaths | $\begin{array}{r} \text { Ratel } \\ 100,000 \\ \hline \end{array}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{aligned} & \text { Rate/ } \\ & 100,000 \\ & \hline \end{aligned}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 111 | 41.44 | 53. | 53. | 1.59 | 1.59 | 99 | 37.64 | 56. | 56. | 1.95 | 1.95 |
| 1-4 | 40 | 5.10 | 28. | 81. | 7.05 | 2.17 | 40 | 4.20 | 19. | 75. | 5.96 | 2.36 |
| 5-9 | 12 | 0.96 | 2. | 82. | 0.95 | 2.11 | 16 | 1.27 | 5. | 81. | 5.32 | 2.45 |
| 10-14 | 23 | 1.71 | 9. | 91. | 10.06 | 2.30 | 22 | 1.68 | 12. | 93. | 17.30 | 2.76 |
| 15-19 | 71 | 4.75 | 38. | 130. | * | 3.26 | 47 | 3.14 | 27. | 120. | * | 3.56 |
| 20-24 | 127 | 9.79 | 80. | 210. | 7.48 | 4.15 | 81 | 5.71 | 52. | 172. | 12.00 | 4.53 |
| 25-29 | 216 | 19.92 | 149. | 358. | 7.30 | 5.06 | 140 | 11.29 | 102. | 274. | 12.57 | 5.95 |
| 30-34 | 357 | 40.99 | 235. | 593. | 9.72 | 6.24 | 202 | 19.88 | 147. | 421. | 14.89 | 7.53 |
| 35-39 | 607 | 91.59 | 350. | 944. | 14.31 | 7.90 | 319 | 40.11 | 232. | 653. | 20.12 | 9.67 |
| 40-44 | 1041 | 183.75 | 502. | 1446. | 18.09 | 9.82 | 570 | 83.27 | 401. | 1054. | 27.43 | 12.84 |
| 45-49 | 1717 | 333.15 | 693. | 2138. | 20.33 | 11.79 | 951 | 151.57 | 641. | 1695. | 32.14 | 16.62 |
| 50-54 | 2706 | 536.41 | 922. | 3060. | 21.30 | 13.62 | 1577 | 252.56 | 986. | 2681. | 36.44 | 20.77 |
| 55-59 | 3866 | 828.71 | 1148. | 4208. | 23.21 | 15.35 | 2326 | 407.87 | 1326. | 4007. | 39.29 | 24.61 |
| 60-64 | 4502 | 1169.11 | 947. | 5155. | 21.57 | 16.21 | 3206 | 660.03 | 1589. | 5596. | 43.78 | 28.10 |
| 65-69 | 5166 | 1557.31 | 478. | 5633. | 13.58 | 15.95 | 4236 | 951.40 | 1639. | 7235. | 44.17 | 30.63 |
| 70-74 | 5154 | 2199.75 | 84. | 5717. | 3.83 | 15.24 | 4882 | 1482.86 | 1443. | 8677. | 40.43 | 31.91 |
| 75-79 | 4540 | 2972.08 | 0. | 5717. | 0.00 | 15.02 | 5095 | 2171.52 | 752. | 9429. | 35.42 | 32.17 |
| 80-84 | 3345 | 4463.36 | 0. | 5717. | * | 15.02 | 4304 | 3442.27 | 60. | 9489. | 7.80 | 31.54 |
| 85-P1 us | 3434 | 6482.78 | 0. | 5717. | * | 15.02 | 5888 | 5557.13 | 0. | 9489. | * | 31.54 |
| All Ages | 37043 | 295.89 | 5717. | -- | 15.02 | * | 34003 | 243.30 | 9489. | -- | 31.54 | * |

[^20]TABLE 53 (Continued)

| Age | $\begin{gathered} 390 \text { to } 398402404 \text { to } 429 / \text { Heart Disease } \\ \text { Race }=\text { Indian } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Desths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 5 | 32.28 | 2. | 2. | 3.57 | 3.57 | 4 | 24.48 | 1. | 1. | 2.45 | 2.45 |
| 1-4 | 2 | 2.81 | 0. | 2. | 1.76 | 3.02 | 3 | 5.25 | 2. | 3. | 7.94 | 4.09 |
| 5-9 | 0 | 0.00 | 0. | 2. | 0.00 | 2.73 | 1 | 0.92 | 0. | 3. | 1.49 | 3.94 |
| 10-14 | 0 | 0.42 | 0. | 2. | 0.00 | 2.41 | 1 | 1.73 | 1. | 4. | 33.83 | 4.74 |
| 15-19 | 2 | 1.93 | 0. | 2. | 0.00 | 1.31 | 2 | 2.78 | 1. | 5. | 5.67 | 4.94 |
| 20-24 | 6 | 8.02 | 3. | 5. | 2.28 | 1.76 | 3 | 3.59 | 1. | 6. | 2.63 | 4.25 |
| 25-29 | 8 | 12.45 | 4. | 9. | 3.00 | 2.13 | 3 | 4.73 | 1. | 7. | 1.98 | 3.63 |
| 30-34 | 9 | 17.17 | 2. | 11. | 1.52 | 2.00 | 6 | 10.34 | 3. | 10. | 4.83 | 3.89 |
| 35-39 | 28 | 68.89 | 12. | 23. | 11.40 | 3.57 | 6 | 13.16 | 0. | 11. | 1.97 | 3.59 |
| 40-44 | 38 | 83.93 | 0. | 23. | 0.00 | 3.10 | 12 | 33.70 | 0. | 14. | 6.10 | 3.96 |
| 45-49 | 45 | 162.30 | 0. | 23. | 0.00 | 2.88 | 14 | 47.53 | 0. | 14. | 0.00 | 3.61 |
| 50-54 | 63 | 253.55 | 0. | 23. | 0.00 | 2.81 | 23 | 87.67 | 0. | 14. | 0.00 | 3.41 |
| 55-59 | 89 | 412.96 | 0. | 23. | * | 2.81 | 33 | 142.29 | 0. | 14. | * | 3.41 |
| 60-64 | 101 | 632.94 | 0. | 23. | * | 2.81 | 49 | 275.47 | 0. | 14. | * | 3.41 |
| 65-69 | 113 | 879.58 | 0. | 23. | * | 2.81 | 75 | 482.87 | 0. | 14. | * | 3.41 |
| 70-74 | 105 | 1182.94 | 0. | 23. | * | 2.81 | 79 | 712.24 | 0. | 14. | * | 3.41 |
| 75-79 | 100 | 1645.08 | 0. | 23. | * | 2.81 | 75 | 974.63 | 0. | 14. | * | 3.41 |
| 80-84 | 72 | 2506.96 | 0. | 23. | * | 2.81 | 80 | 1898.88 | 0. | 14. | * | 3.41 |
| 85-Plus | 90 | 3894.42 | 0. | 23. | * | 2.81 | 113 | 3164.79 | 0. | 14. | * | 3.41 |
| All Ages | 867 | 123.46 | 23. | -- | 2.81 | * | 581 | 80.95 | 14. | -- | 3.41 | * |


| Age | Male Race |  |  |  |  |  | sian |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | ZEx Dth | \%Cumul | Deaths | $\begin{aligned} & \text { Rate/ } \\ & 100,000 \\ & \hline \end{aligned}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 9 | 27.77 | 2. | 2. | * | * | 5 | 17.55 | 0 . | 0. | * | * |
| 1-4 | 4 | 3.13 | 1. | 3. | * | * | 3 | 2.32 | 0 . | 1. | * | * |
| 5-9 | 1 | 0.43 | 0. | 3. | * | * | 1 | 0.90 | 0. | 1. | * | * |
| : 0-14 | 2 | 1.16 | 0. | 3. | * | * | 2 | 1.47 | 1. | 2. | * | * |
| 15-19 | 4 | 2.68 | 1. | 4. | * | * | 1 | 0.96 | 0. | 2. | * | * |
| 20-24 | 8 | 4.88 | 2. | 6. | * | * | 4 | 2.25 | 0. | 2. | * | * |
| 25-29 | 7 | 4.31 | 0. | 6. | * | * | 7 | 3.52 | 1. | 3. | * | * |
| 30-34 | 18 | 10.44 | 0. | 6. | * | * | 9 | 4.29 | 0. | 3. | * | * |
| 35-39 | 24 | 18.24 | 0. | 6. | * | * | 9 | 5.97 | 0. | 3. | * | * |
| 40-44 | 45 | 42.03 | 0. | 6. | * | * | 15 | 13.29 | 0. | 3. | * | * |
| 45-49 | 66 | 80.14 | 0. | 6. | * | * | 22 | 22.36 | 0. | 3. | * | * |
| 50-54 | 115 | 185.18 | 0. | 6. | * | * | 32 | 36.41 | 0. | 3. | * | * |
| 55-59 | 168 | 275.02 | 0. | 6. | * | * | 53 | 77.45 | 0. | 3. | * | * |
| 60-64 | 178 | 394.49 | 0. | 6. | * | * | 79 | 150.66 | 0. | 3. | * | * |
| 65-69 | 240 | 636.59 | 0. | 6. | * | * | 100 | 245.84 | 0 . | 3. | * | * |
| 70-74 | 345 | 1114.02 | 0. | 6. | * | * | 124 | 456.69 | 0. | 3. | * | * |
| 75-79 | 349 | 1780.79 | 0. | 6. | * | * | 172 | 882.41 | 0. | 3. | * | * |
| 80-84 | 256 | 2842.95 | 0. | 6. | * | * | 172 | 1427.98 | 0. | 3. | * | * |
| 85-Plus | 293 | 5631.45 | 0. | 6. | * | * | 302 | 3485.46 | 0. | 3. | * | * |
| All Ages | 2140 | 126.36 | 6. | -- | * | * | 1111 | 61.50 | 3. | -- | * | * |

Mortality, U.S., 1979-1981: Excess Deaths From Ischemic Heart Disease

| Age | 410 to $414 /$ Ischemic Heart Diseage Race * White |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Pemale |  |  |  |  |  |
|  | Deatha | $\begin{array}{r} \text { Ratel } \\ 100,000 \\ \hline \end{array}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deathe | $\begin{array}{r} \text { Rate/ } \\ 100,000 \\ \hline \end{array}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 13 | 0.73 | 0 . | 0. | 999.00 | * | 7 | 0.53 | 0 . | 0 . | * | * |
| 1-4 | 5 | 0.09 | 0. | 0. | * | * | 3 | 0.06 | 0. | 0. | * | * |
| 5-9 | 8 | 0.11 | 0. | 0. | * | * | 5 | 0.08 | 0. | 0. | * | * |
| 10-14 | 4 | 0.06 | 0. | 0. | * | * | 3 | 0.04 | 0. | 0. | * | * |
| 15-19 | 17 | 0.20 | 0. | 0. | * | * | 8 | 0.10 | 0. | 0. | * | * |
| 20-24 | 57 | 0.65 | 0. | 0. | * | * | 22 | 0.25 | 0. | 0. | * | * |
| 25-29 | 184 | 2.30 | 0. | 0. | * | * | 42 | 0.52 | 0. | 0. | * | * |
| 30-34 | 592 | 8.21 | 0. | 0 . | * | * | 122 | 1.66 | 0. | 0. | * | * |
| 35-39 | 1642 | 28.16 | 0. | 0. | * | * | 303 | 5.10 | 0. | 0. | * | * |
| 40-44 | 3586 | 73.94 | 0. | 0. | * | * | 694 | 13.95 | 0. | 0. | * | * |
| 45-49 | 7145 | 158.35 | 0. | 0. | * | * | 1507 | 31.27 | 0. | 0. | * | * |
| 50-54 | 13532 | 281.24 | 0. | 0. | * | * | 3305 | 63.08 | 0. | 0. | * | * |
| 55-59 | 22575 | 465.19 | 0. | 0. | * | * | 6655 | 123.58 | 0 . | 0. | * | * |
| 60-64 | 30972 | 742.18 | 0. | 0. | * | * | 11643 | 242.42 | 0. | 0. | * | * |
| 65-69 | 39463 | 1133.46 | 0. | 0. | * | * | 18814 | 434.44 | 0. | 0. | * | * |
| 70-74 | 44224 | 1732.65 | 0. | 0. | * | * | 27883 | 786.98 | 0. | 0. | * | * |
| 75-79 | 42277 | 2561.64 | 0. | 0. | * | * | 37026 | 1392.00 | 0. | 0 . | * | * |
| 80-84 | 35835 | 3881.15 | 0. | 0. | * | * | 44474 | 2524.00 | 0. | 0. | * | * |
| 85-Plus | 40770 | 6635.67 | 0. | 0. | * | * | 76621 | 5356.01 | 0. | 0. | * | * |
| All Ages | 283407 | 309.11 | 0. | 0 . | * | * | 229135 | 236.99 | 0. | 0. | * | * |

Race - Black
Male Female

| Age | Deatha | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | 7 Cumul |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Under 1 | 2 | 0.75 | 0 . | 0 . | 0.00 | 0.00 | 2 | 0.70 | 1. | 1. | 0.02 | 0.02 |
| 1-4 | 1 | 0.14 | 0. | 0. | 0.11 | 0.01 | 1 | 0.11 | 0. | 1. | 0.13 | 0.03 |
| 5-9 | 1 | 0.08 | 0. | 0. | 0.00 | 0.01 | 1 | 0.11 | 0. | 1. | 0.29 | 0.04 |
| 10-14 | 1 | 0.05 | 0. | 0. | 0.00 | 0.01 | 0 | 0.03 | 0. | 1. | 0.00 | 0.04 |
| 15-19 | 7 | 0.45 | 4. | 4. | * | 0.10 | 4 | 0.27 | 3. | 4. | * | 0.11 |
| 20-24 | 20 | 1.56 | 12. | 16. | 1.11 | 0.32 | 8 | 0.54 | 4. | 8. | 0.94 | 0.21 |
| 25-29 | 59 | 5.44 | 34. | 50. | 1.67 | 0.71 | 22 | 1.75 | 15. | 23: | 1.67 | 0.50 |
| 30-34 | 144 | 16.49 | 72. | 122. | 2.98 | 1.29 | 54 | 5.27 | 37. | 60. | 3.73 | 1.07 |
| 35-39 | 288 | 43.53 | 102. | 224. | 4.16 | 1.87 | 108 | 13.54 | 67. | 127. | 5.83 | 1.88 |
| 40-44 | 561 | 79.08 | 142. | 367. | 5.13 | 2.49 | 247 | 36.08 | 152. | 278. | 16.35 | 3.39 |
| 45-49 | 986 | 191.35 | 170. | 537. | 4.99 | 2.96 | 464 | 73.98 | 268. | 547. | 13.47 | 5.36 |
| 50-54 | 1805 | 318.12 | 186. | 723. | 4.30 | 3.22 | 854 | 136.77 | 460. | 1007 . | 17.00 | 7.80 |
| 55-59 | 2326 | 498.67 | 156. | 879. | 3.16 | 3.21 | 1339 | 234.80 | 634. | 1641. | 16.79 | 10.08 |
| 60-64 | 2736 | 710.64 | 0. | 879. | 0.00 | 2.76 | 1921 | 395.44 | 743. | 2384. | 20.49 | 11.97 |
| 65-69 | 3229 | 973.30 | 0. | 879. | 0.00 | 2.49 | 2581 | 579.72 | 647. | 3031. | 17.43 | 12.83 |
| 70-74 | 3246 | 1385.46 | 0. | 879. | 0.00 | 2.34 | 2977 | 904.17 | 386. | 3417. | 10.81 | 12.57 |
| 75-79 | 2860 | 1872.06 | 0. | 879. | 0.00 | 2.31 | 3160 | 1346.72 | 0. | 3417. | 0.00 | 11.66 |
| 80-84 | 2096 | 2796.94 | 0. | 879. | * | 2.31 | 2662 | 2128.92 | 0. | 3417. | 0.00 | 11.36 |
| 85-Plus | 2124 | 4010.12 | 0. | 879. | * | 2.31 | 3641 | 3436.08 | 0. | 3417. | * | 11.36 |
| All Ages | 22293 | 178.07 | 879. | -- | 2.31 | * | 26045 | 143.43 | 3417. | - | 11.36 | * |

*Percent values are not given when the base of calculation (the exces deaths from all cauges for a particular age, gex, and racial group) is equal to zero.
From: Mortality Rates, Exceas Deatha. National Center for Health Statistica' Death Certificate Data Tapes for 1979, 1980 , and 1981. (Tables aupplied by the DHHS Task Force on Black and Minority Health.)

## TABLE 54 (Continued)

| Age | 410 to $414 /$ Ischemic Heart Disease Race $=$ Indisn |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rste/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rste/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 1-4 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 5-9 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 | 0 | 0.46 | 0. | 0. | 0.20 | 0.34 |
| 10-14 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 | 0 | 0.00 | 0. | 0. | 0.00 | 0.33 |
| 15-19 | 0 | 0.39 | 0. | 0. | 0.22 | 0.10 | 0 | 0.00 | 0. | 0. | 0.00 | 0.26 |
| -0-24 | 1 | 0.89 | 0. | 0. | 0.12 | 0.11 | 0 | 0.00 | 0. | 0. | 0.00 | 0.19 |
| 25-29 | 2 | 2.71 | 0. | 1. | 0.20 | 0.14 | 0 | 0.53 | 0 . | 0. | 0.00 | 0.13 |
| 30-34 | 6 | 11.45 | 2. | 2. | 1.55 | 0.42 | 2 | 3.04 | 1. | 1. | 1.36 | 0.40 |
| 35-39 | 15 | 37.29 | 4. | 6. | 3.45 | 0.93 | 2 | 3.87 | 0. | 1. | 0.00 | 0.34 |
| 40-44 | 18 | 54.31 | 0. | 6. | 0.00 | 0.80 | 4 | 12.17 | 0. | 1. | 0.00 | 0.29 |
| 45-49 | 35 | 126.50 | 0. | 6. | 0.00 | 0.75 | 7 | 24.32 | 0. | 1. | 0.00 | 0.26 |
| 50-54 | 47 | 189.49 | 0. | 6. | 0.00 | 0.73 | 13 | 50.10 | 0. | 1. | 0.00 | 0.25 |
| 55-59 | 68 | 318.25 | 0. | 6. | * | 0.73 | 18 | 78.26 | 0. | 1. | * | 0.25 |
| 60-64 | 75 | 470.54 | 0. | 6. | * | 0.73 | 31 | 174.96 | 0. | 1. | * | 0.25 |
| 65-69 | 85 | 664.23 | 0. | 6. | * | 0.73 | 50 | 325.51 | 0. | 1. | * | 0.25 |
| 70-74 | 79 | 892.86 | 0. | 6. | * | 0.73 | 52 | 473.82 | 0. | 1. | * | 0.25 |
| 75-79 | 70 | 1142.26 | 0. | 6. | * | 0.73 | 44 | 578.69 | 0. | 1. | * | 0.25 |
| 80-84 | 47 | 1636.49 | 0. | 6. | * | 0.73 | 51 | 1218.45 | 0. | 1. | * | 0.25 |
| 85-P1us | 54 | 2322.23 | 0. | 6. | * | 0.73 | 70 | 1975.66 | 0. | 1. | * | 0.25 |
| All Ages | 603 | 85.92 | 6. | -- | 0.73 | * | 347 | 48.36 | -- | 1. | 0.25 | * |

Race $=$ Asisn

| Age | Male |  |  |  |  |  | Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 2 | 5.34 | 1. | 1. | * | * | 0 | 1.10 | 0 . | 0. | * | * |
| 1-4 | 1 | 0.57 | 1. | 2. | * | * | 1 | 0.58 | 1. | 1. | * | * |
| 5-9 | 0 | 0.00 | 0. | 2. | * | * | 0 | 0.22 | 0. | 1. | * | * |
| 10-14 | 0 | 0.00 | 0. | 2. | * | * | 0 | 0.00 | 0. | 1. | * | * |
| 15-19 | 0 | 0.00 | 0. | 2. | * | * | 0 | 0.00 | 0. | 1. | * | * |
| 20-24 | 2 | 1.06 | 1. | 3. | * | * | 0 | 0.20 | 0 . | 1. | * | * |
| 25-29 | 2 | 1.37 | 0. | 3. | * | * | 1 | 0.33 | 0. | 1. | * | * |
| 39-34 | 10 | 6.11 | 0. | 3. | * | * | 2 | 0.82 | 0. | 1. | * | * |
| 35-39 | 14 | 10.39 | 0. | 3. | * | * | 4 | 2.52 | 0. | 1. | * | * |
| 40-44 | 31 | 28.43 | 0. | 3. | * | * | 6 | 5.61 | 0. | 1. | * | * |
| 45-49 | 53 | 64.44 | 0 . | 3. | * | * | 11 | 11.52 | 0. | 1. | * | * |
| 50-54 | 92 | 131.66 | 0. | 3. | * | * | 17 | 19.34 | 0. | 1. | * | * |
| 55-59 | 128 | 208.99 | 0. | 3. | * | * | 33 | 47.44 | 0 . | 1. | * | * |
| 60-64 | 133 | 294.02 | 0. | 3. | * | * | 52 | 99.39 | 0. | 1. | * | * |
| 65-69 | 189 | 442.55 | 0. | 3. | * | * | 70 | 173.49 | 0. | 1. | * | * |
| 70-74 | 264 | 853.54 | 0. | 3. | * | * | 86 | 317.59 | 0. | 1. | * | * |
| 75-79 | 261 | 1331.77 | 0 . | 3. | * | * | 126 | 648.13 | 0. | 1. | * | * |
| 80-84 | 188 | 2086.81 | 0. | 3. | * | * | 130 | 1079.29 | 0. | 1. | * | * |
| 85-Plus | 221 | 4258.87 | 0. | 3. | * | * | 224 | 2584.25 | 0. | 1. | * | * |
| All Age 8 | 1590 | 93.88 | 3. | -- | * | * | 764 | 42.26 | 1. | -- | * | * |

## TABLE 55

Mortality, U.S., 1979-1981: Excess Deaths From Cerebrovascular Disease

## 430 to 438/Cerebrovagcular Disease

 Race -White| Age | Male |  |  |  |  |  | Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deaths | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | 2Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 54 | 3.85 | 0 . | 0. | * | * | 41 | 3.10 | 0 . | 0. | * | * |
| 1-4 | 19 | 0.37 | 0. | 0. | * | * | 18 | 0.38 | 0. | 0. | * | * |
| 5-9 | 19 | 0.29 | 0. | 0. | * | * | 16 | 0.26 | 0. | 0. | * | * |
| 10-14 | 21 | 0.28 | 0. | 0. | * | * | 21 | 0.30 | 0. | 0. | * | * |
| 15-19 | 53 | 0.61 | 0. | 0. | * | * | 46 | 0.55 | 0. | 0. | * | * |
| 20-24 | 105 | 1.21 | 0. | 0. | * | * | 85 | 0.98 | 0. | 0. | * | * |
| 25-29 | 141 | 1.76 | 0. | 0. | * | * | 120 | 1.51 | 0. | 0. | * | * |
| 30-34 | 200 | 2.74 | 0. | 0. | * | * | 198 | 2.70 | 0. | 0. | * | * |
| 35-39 | 288 | 4.94 | 0. | 0. | * | * | 310 | 5.23 | 0. | 0. | * | * |
| 40-44 | 442 | 9.11 | 0. | 0. | * | * | 459 | 9.22 | 0. | 0. | * | * |
| 45-49 | 726 | 15.66 | 0 . | 0. | * | * | 765 | 15.88 | 0. | 0. | * | * |
| 50-54 | 1377 | 27.90 | 0. | 0. | * | * | 1213 | 23.14 | 0. | 0. | * | * |
| 55-59 | 2244 | 46.24 | 0. | 0. | * | * | 1988 | 36.91 | 0. | 0. | * | * |
| 60-64 | 3675 | 88.13 | 0. | 0. | * | * | 3081 | 64.15 | 0. | 0. | * | * |
| 65-69 | 5846 | 167.90 | 0. | 0. | * | * | 5079 | 117.29 | 0. | 0. | * | * |
| 70-74 | 8726 | 341.88 | 0. | 0. | * | * | 8023 | 243.37 | 0. | 0. | * | * |
| 75-79 | 10734 | 650.37 | 0. | 0. | * | * | 13389 | 503.38 | 0. | 0. | * | * |
| 80-84 | 11037 | 1195.41 | 0. | 0. | * | * | 18474 | 1048.42 | 0. | 0. | * | * |
| 85-Plus | 13600 | 2213.46 | 0. | 0. | * | * | 33537 | 2344.36 | 0. | 0. | * | * |
| All Ages | 59307 | 64.69 | 0. | 0. | * | * | 87463 | 90.46 | 0. | 0. | * | * |

Race $=$ Black

| Age | Male |  |  |  |  |  | Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deatha | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | 2Ex Dth | \%Cumul | Deathe | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumu 1 |
| Under 1 | 30 | 11.23 | 20. | 20. | 0.59 | 0.59 | 19 | 7.33 | 11. | 11. | 0.39 | 0.39 |
| 1-4 | 5 | 0.56 | 2. | 22. | 0.46 | 0.58 | 4 | 0.42 | 0. | 12. | 0.13 | 0.36 |
| 5-9 | 4 | 0.32 | 0. | 22. | 0.20 | 0.56 | 4 | 0.30 | 0. | 12. | 0.48 | 0.37 |
| 10-14 | 6 | 0.47 | 3. | 24. | 3.12 | 0.62 | 6 | 0.45 | 2. | 14. | 2.92 | 0.42 |
| 15-19 | 12 | 6.81 | 3. | 27. | * | 0.69 | 13 | 0.87 | 5. | 19. | * | 0.56 |
| 20-24 | 15 | 2.72 | 20. | 47. | 1.84 | 0.93 | 34 | 2.41 | 20. | 39. | 4.68 | 1.03 |
| 25-29 | 52 | 4.83 | 33. | 80. | 1.63 | 1.13 | 65 | 5.28 | 47. | 86. | 5.73 | 1.86 |
| 30-34 | 94 | 10.83 | 76. | 151. | 2.91 | 1.59 | 89 | 8.75 | 62. | 147. | 6.24 | 2.64 |
| 35-39 | 155 | 23.45 | 123. | 273. | 5.01 | 2.29 | 124 | 15.59 | 82. | 230. | 7.16 | 3.41 |
| 40-44 | 229 | 40.36 | 177. | 450. | 6.38 | 3.06 | 196 | 28.58 | 133. | 362. | 9.06 | 4.41 |
| 45-49 | 346 | 67.08 | 265. | 715. | 7.78 | 3.95 | 312 | 49.71 | 212. | 575. | 10.67 | 5.63 |
| 50-54 | 224 | 103.79 | 382. | 1098. | 8.83 | 4.89 | 457 | 73.24 | 313. | 688. | 11.56 | 6.88 |
| 55-59 | 719 | 154.12 | 503. | 1601. | 10.18 | 5.84 | 620 | 108.78 | 410. | 1297. | 12.14 | 7.97 |
| 60-64 | 921 | 239.19 | 582. | 2183. | 13.25 | 6.86 | 805 | 165.64 | 493. | 1790. | 13.59 | 8.99 |
| 65-69 | 1210 | 364.86 | 653. | 2836. | 18.54 | 8.03 | 1208 | 271.37 | 686. | 2477. | 18.49 | 10.48 |
| 70-74 | 1396 | 505.92 | 595. | 3431. | 27.08 | 9.15 | 1590 | 482.81 | 788. | 3265. | 22.09 | 12.01 |
| 75-79 | 1336 | 874.38 | 342. | 3774. | 61.53 | 9.91 | 1699 | 724.22 | 518. | 3783. | 24.41 | 12.91 |
| 80-84 | 992 | 1323.98 | 96. | 3870. | * | 10.16 | 1497 | 1197.37 | 186. | 3969. | 24.16 | 13.19 |
| 85-P1us | 978 | 1845.84 | 0. | 3870. | * | 10.16 | 2007 | 1893.90 | 0. | 3969. | * | 13.19 |
| All Ages | 9045 | 72.25 | 3870. | -- | 10.16 | * | 10750 | 76.92 | 3969. | 0. | 13.19 | * |

[^21]TABLE 55 (Continued)

| Age | $\begin{gathered} 430 \text { to } 438 / \text { Cerebrovaacular Diaease } \\ \text { Race }=\text { Indian } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deatha | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | EEx Dth | \%Cumul |
| Under 1 | 1 | 4.04 | 0. | 0. | 0.06 | 0.06 | 0 | 0.00 | 0 . | 0. | 0.00 | 0.00 |
| 1-4 | 0 | 0.00 | 0. | 0. | 0.00 | 0.04 | 1 | 1.17 | 0. | 0. | 2.01 | 0.60 |
| 5-9 | 0 | 0.00 | 0. | 0. | 0.00 | 0.04 | 0 | 0.00 | 0. | 0. | 0.00 | 0.56 |
| 10-14 | 0 | 0.00 | 0. | 0. | 0.00 | 0.04 | 0 | 0.00 | 0. | 0. | 0.00 | 0.55 |
| 15-19 | 1 | 0.77 | 0. | 0. | 0.19 | 0.11 | 1 | 0.79 | 0. | 1. | 0.97 | 0.64 |
| 20-24 | 1 | 1.34 | 0. | 0. | 0.07 | 0.09 | 1 | 1.35 | 0. | 1. | 0.62 | 0.63 |
| 25-29 | 0 | 0.54 | 0. | 0. | 0.00 | 0.06 | 1 | 1.05 | 0. | 1. | 0.00 | 0.46 |
| 30-34 | 2 | 3.16 | 0. | 0. | 0.21 | 0.09 | 2 | 4.26 | 1. | 2. | 1.54 | 0.69 |
| 35-39 | 1 | 3.24 | 0. | 0. | 0.00 | 0.08 | 3 | 6.19 | 0. | 2. | 0.87 | 0.72 |
| 40-44 | 3 | 8.89 | 0. | 0. | 0.00 | 0.07 | 5 | 13.10 | 1. | 4. | 2.81 | 1.00 |
| 45-49 | 5 | 16.71 | 0. | 1. | 0.50 | 0.10 | 6 | 19.90 | 1. | 5. | 3.48 | 1.22 |
| 50-54 | 8 | 32.03 | 1. | 2. | 4.85 | 0.22 | 5 | 18.79 | 0. | 5. | 6.00 | 1.15 |
| 55-59 | 14 | 63.65 | 4. | 6. | * | 0.67 | 8 | 32.73 | 0. | 5. | * | 1.15 |
| 60-64 | 15 | 93.69 | 1. | 6. | * | 0.77 | 10 | 55.84 | 0. | 5. | * | 1.15 |
| 65-69 | 21 | 160.86 | 0. | 6. | * | 0.77 | 20 | 127.19 | 2. | 6. | * | 1.52 |
| 70-74 | 20 | 222.27 | 0. | 6. | * | 0.77 | 17 | 150.90 | 6. | 6. | * | 1.52 |
| 75-79 | 24 | 398.97 | 0. | 6. | * | 0.77 | 21 | 274.12 | 0. | 6. | * | 1.52 |
| 80-84 | 19 | 649.95 | 0. | 6. | * | 0.77 | 20 | 474.72 | 0. | 6. | * | 1.52 |
| 85-Plua | 30 | 1312.56 | 0. | 6. | * | 0.77 | 43 | 1217.23 | 0. | 6. | * | 1.52 |
| All Agea | 164 | 23.31 | 6. | -- | 0.77 | * | 162 | 22.56 | 6. | -- | 1.52 | * |


| Age | Male |  |  |  |  |  | Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deatha | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deatha | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 1 | 4.27 | 0 . | 0. | * | * | 2 | 6.58 | 1. | 1. | * | * |
| 1-4 | 1 | 0.57 | 0. | 0. | * | * | 1 | 0.58 | 0. | 1. | * | * |
| 5-9 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.22 | 0. | 1. | * | * |
| 00-14 | 0 | 0.00 | 0. | 0. | * | * | 1 | 0.49 | 0. | 2. | * | * |
| 15-19 | 1 | 0.67 | 0. | 0. | * | * | 0 | 0.24 | 0. | 2. | * | * |
| 20-24 | 2 | 1.27 | 0. | 1. | * | * | 1 | 0.41 | 0. | 2. | * | * |
| 25-29 | 2 | 0.98 | 0. | 1. | * | * | 4 | 1.84 | 1. | 2. | * | * |
| 30-34 | 5 | 2.95 | 0. | 1. | * | * | 3 | 1.65 | 0. | 2. | * | * |
| 35-39 | 6 | 4.31 | 0. | 1. | * | * | 6 | 4.36 | 0. | 2. | * | * |
| 40-44 | 8 | 7.11 | 0. | 1. | * | * | 9 | 7.68 | 0. | 2. | * | * |
| 45-49 | 15 | 18.12 | 2. | 3. | * | * | 19 | 18.97 | 3. | 5. | * | * |
| 50-54 | 21 | 30.16 | 2. | 4. | * | * | 22 | 25.41 | 2. | 7. | * | * |
| 55-59 | 31 | 50.20 | 2. | 7. | * | * | 29 | 42.11 | 4. | 11. | * | * |
| 60-64 | 31 | 68.70 | 0. | 7. | * | * | 28 | 53.18 | 0. | 11. | * | * |
| 65-69 | 61 | 156.59 | 0. | 7. | * | * | 39 | 95.38 | 0. | 11. | * | * |
| 70-74 | 80 | 258.32 | 0. | 7. | * | * | 43 | 157.56 | 0. | 11. | * | * |
| 75-79 | 93 | 474.54 | 0. | 7. | * | * | 65 | 331.76 | 0. | 11. | * | * |
| 80-84 | 74 | 819.16 | 0. | 7. | * | * | 82 | 678.01 | 0. | 11. | * | * |
| 85-Plua | 77 | 1431.62 | 0. | 7. | * | * | 125 | 1448.16 | 0. | 11. | * | * |
| All Agea | 508 | 29.98 | 7. | -- | * | * | 478 | 26.43 | 11. | -- | * | * |

TABLE 56
Mortality, U.S., 1979-1981: Excess Deaths From Hypertensive Disease

| Age | $\begin{gathered} 401 \text { to 405/Hypertensive Disease } \\ \text { Race = White } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deatha | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | EEx Dth | \%Cumul |
| Under 1 | 3 | 0.19 | 0 . | 0 . | * | * | 1 | 0.08 | 0 . | 0. | * | * |
| 1-4 | 0 | 0.00 | 0. | 0. | * | * | 1 | 0.02 | 0. | 0. | * | * |
| 5-9 | 2 | 0.02 | 0. | 0. | * | * | 1 | 0.02 | 0. | 0. | * | * |
| 10-14 | 1 | 0.01 | 0. | 0. | * | * | 2 | 0.02 | 0. | 0. | * | * |
| 15-19 | 3 | 0.03 | 0. | 0. | * | * | 2 | 0.02 | 0. | 0. | * | * |
| 20-24 | 7 | 0.08 | 0. | 0. | * | * | 5 | 0.05 | 0. | 0. | * | * |
| 25-29 | 21 | 0.27 | 0. | 0. | * | * | 8 | 0.10 | 0. | 0. | * | * |
| 30-34 | 35 | 0.48 | 0. | 0. | * | * | 17 | 0.23 | 0. | 0. | * | * |
| 35-39 | 59 | 1.02 | 0. | 0. | * | * | 27 | 0.45 | 0. | 0. | * | * |
| 40-44 | 122 | 2.52 | 0. | 0. | * | * | 57 | 1.15 | 0. | 0. | * | * |
| 45-49 | 226 | 4.86 | 0. | 0. | * | * | 125 | 2.59 | 0. | 0. | * | * |
| 50-54 | 444 | 9.03 | 0. | 0. | * | * | 234 | 4.47 | 0. | 0. | * | * |
| 55-59 | 779 | 16.05 | 0. | 0. | * | * | 444 | 8.24 | 0. | 0. | * | * |
| 60-64 | 1029 | 24.67 | 0. | 0. | * | * | 717 | 14.94 | 0. | 0. | * | * |
| 65-69 | 1352 | 38.84 | 0. | 0. | * | * | 1138 | 26.28 | 0. | 0. | * | * |
| 70-74 | 1511 | 59.20 | 0. | 0. | * | * | 1678 | 47.37 | 0. | 0. | * | * |
| 75-79 | 1575 | 75.45 | 0. | 0. | * | * | 2277 | 85.60 | 0. | 0. | * | * |
| 80-84 | 1391 | 150.62 | 0. | 0. | * | * | 2775 | 157.47 | 0. | 0. | * | * |
| 85-P1us | 1708 | 278.05 | 0. | 0. | * | * | 4529 | 316.61 | 0. | 0. | * | * |
| All Ages | 10269 | 11.20 | 0. | 0. | * | * | 14038 | 14.52 | 0. | 0. | * | * |

Race $=$ Black

|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Age | Deaths | $\begin{gathered} \text { Ratel } \\ 100.000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumu I |
| Under 1 | 0 | 0.12 | 0. | 0 . | 0.00 | 0.00 | 0 | 0.13 | 0 . | 0. | 0.00 | 0.00 |
| 1-4 | 1 | 0.07 | 1. | 1. | 0.17 | 0.02 | 1 | 0.11 | 1. | 1. | 0.29 | 0.03 |
| 5-9 | 0 | 0.00 | 0. | 1. | 0.00 | 0.02 | 0 | 0.00 | 0. | 1. | 0.00 | 0.03 |
| 10-14 | 1 | 0.05 | 1. | 1. | 0.66 | 0.03 | 1 | 0.05 | 0. | 1. | 0.50 | 0.04 |
| 15-19 | 3 | 0.18 | 2. | 3. | * | 0.08 | 1 | 0.09 | 1. | 2. | * | 0.07 |
| 20-24 | 6 | 0.49 | 5. | 7. | 0.49 | 0.17 | 6 | 0.40 | 5. | 7. | 1.13 | 0.19 |
| 25-29 | 23 | 2.12 | 20. | 29. | 0.99 | 0.41 | 20 | 1.62 | 19. | 26. | 2.29 | 0.56 |
| 30-34 | 49 | 5.63 | 45. | 73. | 1.85 | 0.77 | 31 | 3.65 | 29. | 55. | 2.90 | 0.98 |
| 35-39 | 78 | 11.83 | 72. | 145. | 2.93 | 1.21 | 57 | 7.21 | 54. | 108. | 4.67 | 1.61 |
| 40-44 | 147 | 26.01 | 133. | 278. | 4.79 | 1.89 | 107 | 15.63 | 99. | 207. | 6.77 | 2.59 |
| 45-49 | 205 | 39.85 | 180. | 458. | 5.29 | 2.53 | 177 | 28.20 | 161. | 308. | 8.07 | 3.61 |
| 50-54 | 317 | 42.76 | 271. | 729. | 6.26 | 3.25 | 276 | 44.26 | 248. | 617. | 9.18 | 4.78 |
| 55-59 | 413 | 88.60 | 338. | 1068. | 6.85 | 3.90 | 357 | 62.54 | 310. | 926. | 9.18 | 5.69 |
| 60-64 | 436 | 113.32 | 341. | 1409. | 7.78 | 4.43 | 429 | 88.24 | 356. | 1282. | 9.61 | 6.44 |
| 65-69 | 487 | 136.81 | 358. | 1767. | 10.16 | 5.00 | 553 | 124.12 | 436. | 1718. | 11.74 | 7.27 |
| 70-74 | 456 | 193.75 | 318. | 2085. | 14.45 | 5.56 | 584 | 177.47 | 428. | 2146. | 12.00 | 7.89 |
| 75-79 | 365 | 238.73 | 219. | 2304. | 39.36 | 6.05 | 573 | 244.34 | 372. | 2519. | 17.54 | 6.59 |
| 80-84 | 243 | 323.77 | 130: | 2434. | * | 6.39 | 429 | 343.40 | 232. | 2751. | 30.18 | 9.15 |
| 85-Plus | 227 | 427.95 | 79. | 2513. | * | 6.60 | 495 | 467.50 | 160. | 2911. | * | 9.68 |
| All Agea | 3457 | 27.62 | 2513. | -- | 6.60 | * | 4098 | 29.32 | 2911. | -- | 9.68 | * |

*Percent values are not given when the base of calculation (the excesa deatha from all causes for a particular age, aex,
and racial group) is equal to zero.
From: Mortality Rates, Excesa Deatha. National Center for Health Statiatics" Death Certificate Data Tapea for 1979 , 1980 , and 1981. (Tables aupplied by the DHHS Task Force on Black and Minority Health.)

TABLE 56 (Continued)

| Age | $\begin{gathered} 401 \text { to 405/Hypertengive Disease } \\ \text { Race }=\text { Indian } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{aligned} & \text { Rate/ } \\ & 100,000 \\ & \hline \end{aligned}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 0.00 | 0 . | 0. | 0.00 | 0.00 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 1-4 | 0 | 0.56 | 0. | 0. | 1.59 | 0.48 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 5-9 | 0 | 0.00 | 0. | 0. | 0.00 | 0.43 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 10-14 | 0 | 0.00 | 0. | 0. | 0.00 | 0.38 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 15-19 | 0 | 0.00 | 0. | 0. | 0.00 | 0.21 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 20-24 | 1 | 0.89 | 1. | 1. | 0.42 | 0.31 | 0 | 0.45 | 0. | 0. | 0.67 | 0.20 |
| 25-29 | 0 | 0.54 | 0. | 1. | 0.13 | 0.26 | 0 | 0.00 | 0. | 0. | 0.00 | 0.15 |
| 30-34 | 0 | 0.00 | 0. | 1. | 0.00 | 0.20 | 0 | 0.61 | 0. | 0. | 0.37 | 0.19 |
| 35-39 | 1 | 1.62 | 0. | 1. | 0.23 | 0.21 | 1 | 1.55 | 0. | 1. | 1.00 | 0.32 |
| 40-44 | 1 | 2.96 | 0. | 2. | 0.15 | 0.20 | 0 | 0.94 | 0. | 1. | 0.00 | 0.27 |
| 45-49 | 2 | 8.35 | 1. | 2. | 1.66 | 0.31 | 1 | 4.42 | 1. | 2. | 1.58 | 0.39 |
| 50-54 | 2 | 8.01 | 0. | 2. | 0.00 | 0.30 | 1 | 5.01 | 0. | 2. | 0.60 | 0.40 |
| 55-59 | 3 | 15.52 | 0. | 2. | * | 0.30 | 1 | 5.69 | 0. | 2. | * | 0.40 |
| 60-64 | 3 | 16.66 | 0. | 2. | * | 0.30 | 3 | 16.75 | 0. | 2. | * | 0.48 |
| 65-69 | 3 | 25.95 | 0. | 2. | * | 0.30 | 3 | 19.40 | 0. | 2. | * | 0.48 |
| 70-74 | 4 | 48.98 | 0. | 2. | * | 0.30 | 5 | 48.29 | 0. | 2. | * | 0.50 |
| 75-79 | 3 | 54.65 | 0. | 2. | * | 0.30 | 3 | 39.16 | 0. | 2. | * | 0.50 |
| 80-84 | 3 | 92.85 | 0. | 2. | * | 0.30 | 3 | 79.12 | 0. | 2. | * | 0.50 |
| 85-P1ua | 3 | 115.39 | 0. | 2. | * | 0.30 | 5 | 149.81 | 0. | 2. | * | 0.50 |
| All Agea | 30 | 4.22 | 2. | -- | 0.30 | * | 29 | 3.99 | 2. | -- | 0.50 | * |
|  |  | Race $=$ Aatan |  |  |  |  |  |  |  |  |  |  |
| Age | Deaths | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deathe | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 0.00 | 0. | 0. | * | * | 0 | 1.10 | 0 . | 0. | * | * |
| 1-4 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 0. | * | * |
| j-9 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.22 | 0. | 1. | * | * |
| 10-14 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 1. | * | * |
| 15-19 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 1. | * | * |
| 20-24 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 1. | * | * |
| 25-29 | 0 | 0.20 | 0. | 0. | * | * | 0 | 0.17 | 0. | 1. | * | * |
| 30-34 | 1 | 0.39 | 0. | 0. | * | * | 1 | 0.33 | 0. | 1. | * | * |
| 35-39 | 2 | 1.52 | 1. | 1. | * | * | 0 | 0.23 | 0. | 1. | * | * |
| 40-44 | 4 | 3.40 | 1. | 2. | * | * | 2 | 1.48 | 0. | 1. | * | * |
| 45-49 | 4 | 5.24 | 0. | 2. | * | * | 4 | 3.73 | 1. | 2. | * | * |
| 50-54 | 5 | 7.15 | 0. | 2. | * | * | 3 | 3.79 | 0. | 2. | * | * |
| 55-59 | 11 | 18.01 | 1. | 3. | * | * | 5 | 6.78 | 0. | 2. | * | * |
| 60-64 | 9 | 19.95 | 0. | 3. | * | * | 5 | 9.50 | 0. | 2. | * | * |
| 65-69 | 14 | 35.74 | 0. | 3. | * | * | 6 | 15.62 | 0. | 2. | * | * |
| 70-74 | 19 | 60.25 | 0. | 3. | * | * | 8 | 28.31 | 0. | 2. | * | * |
| 75-79 | 18 | 90.15 | 0. | 3. | * | * | 16 | 82.68 | 0. | 2. | * | * |
| 80-84 | 13 | 148.26 | 0. | 3. | * | * | 14 | 116.23 | 0. | 2. | * | * |
| 85-P1ue | 9 | 179.59 | 0. | 3. | * | * | 21 | 242.63 | 0. | 2. | * | * |
| All Ages | 109 | 6.44 | 3. | -- | * | * | 85 | 4.72 | 2. | -- | * | * |

TABLE 57
Mortality, U.S., 1979-1981: Excess Deaths From Diabetes Mellitus

| Age | $\begin{aligned} & \text { 250/D1abetea Mellitus } \\ & \text { Race }=\text { White } \end{aligned}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 3 | 0.21 | 0. | 0. | * | * | 1 | 0.05 | 0. | 0. | * | * |
| 1-4 | 3 | 0.04 | 0. | 0. | * | * | 5 | 0.10 | 0. | 0. | * | * |
| 5-9 | 5 | 0.08 | 0. | 0. | * | * | 6 | 0.09 | 0. | 0. | * | * |
| 10-14 | 7 | 0.10 | 0. | 0. | * | * | 10 | 0.14 | 0. | 0. | * | * |
| 15-19 | 13 | 0.15 | 0. | 0. | * | * | 20 | 0.24 | 0. | 0. | * | * |
| 20-24 | 41 | 0.47 | 0. | 0. | * | * | 38 | 0.44 | 0. | 0. | * | * |
| 25-29 | 91 | 1.14 | 0. | 0. | * | * | 79 | 0.94 | 0. | 0. | * | * |
| 30-34 | 151 | 2.07 | 0. | 0. | * | * | 110 | 1.50 | 0. | 0. | * | * |
| 35-39 | 176 | 3.81 | 0. | 0. | * | * | 123 | 2.07 | 0. | 0. | * | * |
| 40-44 | 211 | 4.35 | 0. | 0. | * | * | 153 | 3.08 | 0. | 0. | * | * |
| 45-49 | 303 | 6.54 | 0. | 0. | * | * | 242 | 5.02 | 0. | 0. | * | * |
| 50-54 | 547 | 11.13 | 0. | 0. | * | * | 452 | 8.63 | 0. | 0. | * | * |
| 55-59 | 907 | 18.68 | 0. | 0. | * | * | 840 | 15.60 | 0. | 0. | * | * |
| 60-64 | 1319 | 31.61 | 0. | 0. | * | $\star$ | 1308 | 27.24 | 0. | 0. | * | * |
| 65-69 | 1677 | 48.17 | 0. | 0. | * | * | 1927 | 44.50 | 0. | 0. | * | * |
| 70-74 | 1920 | 75.23 | 0. | 0. | * | * | 2478 | 69.95 | 0. | 0. | * | * |
| 75-79 | 1820 | 110.30 | 0. | 0. | * | * | 2824 | 106.17 | 0. | 0. | * | * |
| 80-84 | 1461 | 158.20 | 0. | 0. | * | * | 2722 | 154.48 | 0. | 0. | * | * |
| 85-Plus | 1318 | 214.57 | 0. | 0. | * | * | 3126 | 218.52 | 0. | 0. | * | * |
| All Agea | 11973 | 13.06 | 0. | 0. | * | * | 16463 | 17.03 | 0. | 0. | * | * |

Race $=$ Black

| Age | Male Race |  |  |  |  |  | K._ Female |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deatha | $\begin{gathered} \text { Rate/ } \\ 100.000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 | 0 | 0.13 | 0. | 0. | 0.01 | 0.01 |
| 1-4 | 2 | 0.24 | 2. | 2. | 0.49 | 0.05 | 0 | 0.04 | 0. | 0. | 0.00 | 0.01 |
| 5-9 | 1 | 0.11 | 0. | 2. | 0.18 | 0.06 | 1 | 0.11 | 0. | 0. | 9.23 | 0.01 |
| 10-14 | 2 | 0.17 | 1. | 3. | 1.21 | 0.08 | 4 | 0.33 | 3. | 3. | 3.59 | 0.09 |
| 15-19 | 5 | 0.34 | 3. | 6. | *. | 0.15 | 4 | 0.29 | 1. | 4. | * | 0.11 |
| 20-24 | 14 | 1.10 | 8. | 14. | 0.77 | 0.28 | 13 | 0.89 | 6. | 10. | 1.48 | 0.27 |
| 15-29 | 22 | 2.00 | 9. | 24. | 0.46 | 0.33 | 23 | 1.86 | 11. | 21. | 1.32 | 0.45 |
| 30-34 | 37 | 4.21 | 19. | 42. | 0.77 | 0.44 | 28 | 2.72 | 12. | 33. | 1.26 | 0.60 |
| 35-39 | 45 | 7.30 | 28. | 71. | 1.16 | 0.59 | 48 | 6.08 | 32. | 65. | 2.77 | 0.97 |
| 40-44 | 70 | 12.36 | 45. | 116. | 1.63 | 0.79 | 63 | 9.25 | 42. | 107. | 2.89 | 1.31 |
| 45-49 | 95 | 18.44 | 61. | 177. | 1.80 | 0.98 | 107 | 17.10 | 76. | 183. | 3.61 | 1.60 |
| 50-54 | 141 | 27.88 | 85. | 262. | 1.95 | 1.17 | 208 | 33.26 | 154. | 337. | 5.68 | 2.61 |
| 55-59 | 217 | 46.59 | 130. | 392. | 2.63 | 1.43 | 308 | 53.95 | 219. | 556. | 6.88 | 3.41 |
| 60-64 | 236 | 61.81 | 116. | 508. | 2.65 | 1.60 | 425 | 87.42 | 292. | 848. | 8.06 | 4.26 |
| 65-69 | 298 | 89.83 | 138. | 640. | 3.92 | 1.83 | 554 | 124.34 | 356. | 1204. | 9.58 | 5.10 |
| 70-74 | 269 | 114.94 | 93. | 740. | 4.23 | 1.97 | 509 | 166.64 | 318. | 1522. | 8.92 | 5.60 |
| 75-79 | 240 | 162.35 | 80. | 819. | 14.30 | 2.15 | 465 | 198.17 | 216. | 1738. | 10.17 | 5.93 |
| 80-84 | 133 | 177.00 | 14. | 833. | * | 2.19 | 320 | 255.68 | 127. | 1864. | 16.42 | 6.20 |
| 85-plus | 109 | 200.82 | 0. | 833. | * | 2.19 | 322 | 303.91 | 90. | 1955. | * | 6.50 |
| All Agea | 1950 | 15.58 | 833. | -- | 2.19 | * | 3442 | 24.83 | 1955. | -- | 8.50 | * |

*Percent valuea are not given when the bage of calculation (the excesa deatha from all cauges for a particular age, gex, and racial group) is equal to zero.
From: Mortality Rates, Excess Deathe. National Center for Health Statiatics' Death Certificate Data Tapes for 1979 , 1980 , and 1981. (Tableg supplied by the DHHS Task Force on Black and Minority Health.)

TABLE 57 (Continued)

| Age | $\begin{gathered} \text { 250/Diabetes Mellitus } \\ \text { Race }=\text { Indian } \end{gathered}$ |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male |  |  |  |  |  | Female |  |  |  |  |  |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul | Deaths | $\begin{gathered} \text { Ratel } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 2.02 | 0 . | 0. | 0.61 | 0.61 | 0 | 0.00 | 0. | 0. | 0.00 | 0.00 |
| 1-4 | 0 | 0.00 | 0. | 0. | 0.00 | 0.43 | 0 | 0.58 | 0. | 0. | 1.23 | 0.36 |
| 5-9 | 0 | 0.00 | 0. | 0. | 0.00 | 0.38 | 0 | 0.00 | 0. | 0. | 0.00 | 0.34 |
| 10-14 | 0 | 0.00 | 0. | 0. | 0.00 | 0.34 | 0 | 0.00 | 0. | 0. | 0.00 | 0.34 |
| 15-19 | 0 | 0.00 | 0. | 0. | 0.00 | 0.18 | 0 | 0.00 | 0. | 0. | 0.00 | 0.27 |
| 20-24 | 1 | 0.89 | 0. | 1. | 0.22 | 0.20 | 0 | 0.00 | 0. | 0. | 0.00 | 0.19 |
| 25-29 | 2 | 3.25 | 1. | 2. | 1.01 | 0.44 | 1 | 1.05 | 0. | 0. | 0.07 | 0.16 |
| 30-34 | 1 | 1.91 | 0. | 2. | 0.00 | 0.35 | 1 | 1.22 | 0. | 0. | 0.00 | 0.12 |
| 35-39 | 2 | 4.05 | 0. | 2. | 0.39 | 0.36 | 1 | 3.10 | 0. | 1. | 0.93 | 0.25 |
| 40-44 | 6 | 16.79 | 4. | 7. | 4.26 | 0.87 | 1 | 3.74 | 0. | 1. | 0.45 | 0.28 |
| 45-49 | 5 | 16.71 | 3. | 9. | 4.84 | 1.16 | 5 | 16.58 | 3. | 4. | 10.01 | 1.14 |
| 50-54 | 9 | 34.70 | 6. | 15. | 28.34 | 1.84 | 7 | 27.55 | 5. | 10. | 21.17 | 2.29 |
| 55-59 | 10 | 48.13 | 6. | 22. | * | 2.60 | 13 | 56.92 | 10. | 19. | * | 4.61 |
| 60-64 | 10 | 62.46 | 5. | 27. | * | 3.19 | 18 | 102.37 | 13. | 33. | * | 7.84 |
| 65-69 | 11 | 88.22 | 5. | 32. | * | 3.81 | 22 | 142.28 | 15. | 48. | * | 11.47 |
| 70-74 | 13 | 143.16 | 6. | 38. | * | 4.54 | 12 | 105.63 | 4. | 52. | * | 12.42 |
| 75-79 | 8 | 131.17 | 1. | 39. | * | 4.69 | 14 | 178.39 | 6. | 57. | * | 13.75 |
| 80-84 | 5 | 162.49 | 0. | 39. | * | 4.71 | 9 | 221.54 | 3. | 60. | * | 14.43 |
| 85-Plus | 3 | 144.24 | 0. | 39. | * | 4.71 | 4 | 121.72 | 0. | 60. | * | 14.43 |
| All Ages | 85 | 12.10 | 39. | -- | 4.71 | * | 109 | 15.22 | 60. | -- | 14.43 | * |


| Age | Male |  |  |  |  |  | $\ldots$ Pemale |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumu1 | ZEx Dth | \%Cumul | Deaths | $\begin{gathered} \text { Rate/ } \\ 100,000 \\ \hline \end{gathered}$ | Ex Dth | Cumul | \%Ex Dth | \%Cumul |
| Under 1 | 0 | 0.00 | 0 . | 0. | * | * | 0 | 0.00 | 0 . | 0 . | * | * |
| 1-4 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 0. | * | * |
| 5-9 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.22 | 0. | 0. | * | * |
| 10-14 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 0. | * | * |
| 15-19 | 0 | 0.00 | 0. | 0. | * | * | 0 | 0.00 | 0. | 0. | * | * |
| 20-24 | 0 | 0.21 | 0. | 0. | * | * | 1 | 0.41 | 0. | 0. | * | * |
| 25-29 | 0 | 0.20 | 0. | 0. | * | * | 0 | 0.17 | 0. | 0. | * | * |
| 30-34 | 0 | 0.00 | 0. | 0. | * | * | 1 | 0.33 | 0. | 0. | * | * |
| 35-39 | 0 | 0.25 | 0. | 0. | * | * | 0 | 0.00 | 0. | 0. | * | * |
| 40-44 | 4 | 3.71 | 0. | 0. | * | * | 1 | 1.18 | 0. | 0. | * | * |
| 45-49 | 5 | 5.64 | 0. | 0. | * | * | 3 | 3.05 | 0. | 0. | * | * |
| 50-54 | 7 | 10.53 | 0. | 0. | * | * | 5 | 6.07 | 0. | 0. | * | * |
| 55-59 | 11 | 17.46 | 0. | 0. | * | * | 8 | 11.13 | 0. | 0. | * | * |
| 60-64 | 11 | 25.12 | 0. | 0. | * | * | 12 | 22.79 | 0. | 0. | * | * |
| 65-69 | 13 | 34.04 | 0. | 0. | * | * | 15 | 37.00 | 0. | 0. | * | * |
| 70-74 | 21 | 67.81 | 0. | 0. | * | * | 15 | 54.16 | 0. | 0. | * | * |
| 75-79 | 14 | 73.55 | 0. | 0. | * | * | 16 | 80.37 | 0. | 0. | * | * |
| 80-84 | 12 | 129.73 | 0. | 0. | * | * | 15 | 127.30 | 0. | 0. | * | * |
| 85-Plus | 12 | 224.49 | 1. | 1. | * | * | 18 | 211.82 | 0. | 0. | * | * |
| All Ages | 115 | 6.79 | 1. | -- | * | * | 110 | 0.11 | -- | -- | * | * |

Rank Order and Propartional Mortality (P.M.) of the Ten Leading Causes Of Death, According to Specified Race: United States, 1980

| 10 Leading Causes, United States | ICO-9 Codes | White |  | Chineae |  | Japanese |  | Pilipino |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Rank | P.M. | Rank | P.M. | Rank | P.M. |  |  | P.M. |
| 1. Heart Disease | 390-398, 402, 404-429 | 1 | 39.3 | 1 | 31.8 | 13 | 30.4 | 1 |  |  |
| 2. Cancer | 140-208 | 2 | 21.3 | 22 | 27.4 | 22 | 25.4 | 2 |  |  |
| 3. Cerebrovascular Disease | 430-438 | 3 | 8.6 | 3 | 8.6 | 31 | 11.2 | 3 |  |  |
| 4. Accidents | E800-E949 | 4 | 5.2 | 4 | 4.2 | 4 | 5.4 | 4 |  |  |
| 5. Chronic Ohatructive Pulm. Disease | 490-496 | 5 | 3.0 | 6 | 2.4 | 8 | 2.0 | 6 |  |  |
| 6. Pnellmonia and Influenza | 480-487 | 6 | 2.6 | 5 | 3.0 | 5 | 3.5 | 5 |  |  |
| 7. Diabetes Mellitus | 250 | 7 | 1.7 | 8 | 2.1 | 7 | 2.0 | 7 |  |  |
| 8. Chronlc Llver 6 Cirrhosis Disease | 571 | 8 | 1.4 | 9 | 1.2 | 9 | 1.2 | 9 |  |  |
| 9. Atherosclerosis | 440 | 9 | 1.5. | 10 | 0.9 | 10 | 1.0 | 10 |  |  |
| 10. Suicide 6 Self-inflicted injury | E950-E959 | 10 | 1.5 | 7 | 2.2 | 6 | 2.3 | 8 |  |  |

Source: National Center for Health Statistics

[^22]
# Age-Adjusted Race-Mortality Ratios for Specified Cause of Death: United States, 1980 

| Causes of Death | Chineae | Japaneae | Pilipino |
| :---: | :---: | :---: | :---: |
| lleart Disesse | 0.54 | 0.42 | 0.42 |
| Cancer | 0.76 | 0.60 | 0.40 |
| Cerehrovascular Disease | 0.76 | 0.76 | 0.66 |
| Aceidents | 0.34 | 0.44 | 0.39 |
| Chronic Ohat ructive Pulmonary Disease | 0.50 | 0.34 | 0.31 |
| Preumonia and Influenza | 0.81 | 0.73 | 0.59 |
| Diabetee Mellitus | 0.81 | 0.64 | 0.49 |
| Chronic Liver Diaesae and Cirrhoais | 0.42 | 0.34 | 0.29 |
| Atherocclerosia | 0.57 | 0.41 | 0.25 |
| 8uicide and self-inflicted injury | 0.64 | 0.62 | 0.30 |

Note: Ratios are calculated for each specific cause of death by dividing the age-adjusted death rate of a specified ethnic group by the ageadjusted death rate of the white population

Source: National Center for Health Statistics, published and unpublished data

TABLE 60
Within-Group Sex-Mortality Ratios(1) For All Causes of Death:

United States, 1980

| Age | White | Chinese | Japanese | Pilipino |
| :---: | :---: | :---: | :---: | :---: |
| All ages, erude | 1.23 | 1.63 | 1.33 | 3.25 |
| Age-adjusted ${ }^{2}$ | 1.82 | 1.75 | 1.65 | 1.96 |
| Onder 5 years | 1.28 | 1.13 | 1.37 | 1.01 |
| 5-14 yeara | 1.56 | 0.73 | 1.31 | 1.39 |
| 15-24 years | 3.00 | 2.39 | 2.05 | 2.80 |
| 25-34 years | 2.61 | 1.60 | 1.84 | 1.87 |
| 35-44 jears | 1.86 | 1.27 | 1.17 | 1.38 |
| 45-54 years | 1.88 | 1.70 | 1.64 | 1.34 |
| 55-64 years | 1.98 | 1.94 | 1.72 | 2.18 |
| 65-74 years | 1.96 | 1.97 | 1.95 | 2.58 |
| 75-84 years | 1.65 | 1.86 | 1.60 | 2.07 |
| 85 years and over | 1.28 | 1.31 | 1.38 | 1.71 |

(1) Excludes deaths of nonresidents of the United States. Ratios are computed for each ethnic group by dividing the age-specific death rate of males by the death rates of females in that age-group
(2) Age-adjusted by the direct method, using as the standard population the age distribution of the total population of the United States in 1940. Adjustment is based on ten age-groups

Source: National Center for Health Statistics, published and unpublished data computed by the authors

[^23]| Rank | Non-Spanishsurnamed* | Spanishsurnamed* | Black | Asian-Americans |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  |  | Japanese | Chinese | Korean |
| 1 | Diseases of heart $(42)^{+}$ | Diseases of heart (27) | Diseases of heart (31) | Diseases of heart (32) | Diseases of heart (32) | Malignant neoplasms (25) |
| 2 | $\begin{aligned} & \text { Malignant } \\ & \text { neoplasms } \\ & (22) \end{aligned}$ | Malignant neoplasms (18) | Malignant neoplasms (21) | Malignant neoplasms (22) | Malignant neoplasms (27) | Diseases of heart (16) |
| 3 | Cerebrovascular diseases <br> (9) | Accidents (9) | Cerebrovascular diseases <br> (8) | ```Cerebrovascu- lar diseases (15)``` | ```Cerebrovascu- lar diseases (11)``` | ```Cerebrovascu- lar diseases (10)``` |
| 4 | Accidents (4) | ```Cerebrovascu- lar diseases (6)``` | Accidents (6) | Accidents (5) | Accidents (5) | Accidents (8) |
| 5 | Pneumonia and influenza (3) | Chronic liver disease and cirrhosis (5) | Chronic liver disease and cirrhosis (3) | Pneumonia and influenza <br> (4) | Suicide (4) | Suicide (4) |

[^24][^25] American Heart Association. Greater Los Angeles Affiliate, Inc., 1983

TABLE 62
Age- and Sex-Adjusted Mortality Rates By Race-Ethnicity, Los Angeles County, 1980

| Cause of Death | Age- and sex-adfusted* mortality rates per 100,000 population |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Los Angeles |  | Black | Hispanic $\dagger$ | Asian and Pacific Islanders |  |  |  |
|  | County | White |  |  | Japaneae | Chinese | Filipino | Korean |
| All causes | 819.9 | 870.2 | 1038.3 | 814.8 | 482.5 | 362.8 | 137.2 | 421.8 |
| Major cardiovascular diseases | 409.4 | 429.5 | 472.0 | 390.6 | 255.3 | 157.0 | 84.2 | 143.8 |
| Diseasea of heart | 313.4 | 331.2 | 353.4 | 307.8 | 161.7 | 99.2 | 57.8 | 82.1 |
| Total IHD | 194.0 | 207.7 | 192.5 | 177.7 | 106.9 | 47.4 | 31.4 | 63.9 |
| MI and other acute IHD | 90.9 | 97.9 | 88.3 | 82.3 | 55.4 | 28.1 | 18.8 | 13.1 |
| Chronic IHD | 103.1 | 109.8 | 104.3 | 95.4 | 51.4 | 19.3 | 12.6 | 50.9 |
| Hypertensive disease | 21.0 | 19.5 | 47.4 | -- | -- | -- | -- | -- |
| Cerebrovascular diseases | 74.3 | 75.8 | 94.2 | 63.5 | 79.6 | 48.7 | 19.7 | 48.3 |

*Direct method of adjustment with Los Angelea County population, 1980, as standard.
†Census tracts in which 75 percent or more of the population are persona of Spanish/Bispanic origin or descent.

[^26] Association, Greater Loa Angelea Affiliate, Inc., 1983

## TABLE 63

Annual Mortality Rate From Diseases of the Heart, By Sex, Among Asian And Pacific Islander Groups, Los Angeles County, 1980

| Sos | $4{ }^{\circ}$ | Deatan per 100,000 popoletioz |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Jepeoente | Ca18000000 | 11110100 | Coreas |
| Male |  |  |  |  |  |
|  | <35 | 3.3 | 3.1 | 10.4 | 0.0 |
|  | 35-44 | 29.3 | 0.0 | 13.4 | 17.5 |
|  | 45-54 | 103.4 | 10.0 | 75.2 | 35.4 |
|  | 55-64 | 329.7 | 116.9 | 212.0 | 234.0 |
|  | 65-74 | 469.5 | 651.2 | 255.3 | 420.8 |
|  | 275 | 3064.1 | 1368.2 | 676.2 | 1492.5 |
| Totel mele |  | 165.6 | 68.0 | 61.4 | 37.5 |
| Fasel* |  |  |  |  |  |
|  | <35 | 6.1 | 0.0 | 6.3 | 0.0 |
|  | 35-44 | 24.6 | 16.1 | 0.0 | 0.0 |
|  | 45-54 | 30.6 | 0.0 | 44.2 | 0.0 |
|  | 55-64 | 123.4 | 150.4 | 26.3 | 61.7 |
|  | 65-74 | 364.5 | 263.7 | 200.0 | 244.9 |
|  | 275 | 2375.6 | 1469.1 | 494.2 | 1028.3 |
| fotel fasale |  | 149.0 | 59.8 | 25.1 | 25.3 |
| ```Totale - mele enc fancle``` |  | 156.2 | 62.9 | 42.4 | 31.3 |

From: "Cardiovascular Diseases in Los Angeles", Chapman, J.M. et al. Los Angeles, CA. American Heart Association, Greater Los Angeles Affiliate, Inc., 1983

## TABLE 64

Annual Mortality Rate From Total Ischemic Heart Disease, By Sex, Among Asian and Pacific Islander Groups, Los Angeles County, 1980

| 308 | As* | Deathe per 100,000 populetion |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Japaseeo | Ch100ee | F1118100 | Soreen |
| Mele |  |  |  |  |  |
|  | <35 | 0.0 | 0.0 | 0.0 | 0.0 |
|  | 35-44 | 29.3 | 0.0 | 0.0 | 17.5 |
|  | -5-54 | 25.6 | 0.0 | 50.1 | 0.0 |
|  | 55-54 | 157.7 | 59.2 | 127.2 | 155.0 |
|  | 65-74 | 384.1 | 372.1 | 827.6 | 140.3 |
|  | 275 | 2001.3 | 590.5 | 375.9 | 1452.5 |
| Total |  | 98.1 | 38.2 | 29.9 | 24.1 |
| Peesle |  |  |  |  |  |
|  | <35 | 3.4 | 0.0 | 0.0 | 0.0 |
|  | 35-44 | 0.0 | 15.8 | 0.0 | 0.0 |
|  | 15-54 | 0.0 | 0.0 | 44.2 | 0.0 |
|  | 55-54 | 54.5 | 30.1 | 0.0 | 0.0 |
|  | 55-74 | 255.0 | 131.9 | 120.0 | 163.3 |
|  | 275 | 1772.2 | 587.7 | 329.5 | 1025.3 |
| Tetel feeste |  | 38.6 | 23.6 | 13.5 | 19.0 |
| ```Tarale - 010 esd fansle``` |  | 98.7 | 30.5 | 38.2 | 81.4 |

From: "Cardiovascular Diseases in Los Angeles", Chapman, J.M. et al. Los Angeles, CA. American Heart Association, Greater Los Angeles Affiliate, Inc., 1983

## TABLE 65

Age group $\frac{\text { Japan }^{a}}{\operatorname{Rate} / 1,000} \quad \frac{\text { Hawaii }^{\mathrm{b}}}{\mathrm{N}} \frac{\text { Rate/l,000 }}{\mathrm{N}} \frac{\text { Californiac }}{\text { Rate } / 1,000} \quad$| Significant |
| :---: |
| leveld |

## Definite cases only

| $45-49$ | 0 | -- | 0 | -- | 2 | 2.7 | NS |
| :---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: |
| $50-54$ | 5 | 20.3 | 14 | 4.9 | 4 | 7.6 | $<0.02$ |
| $55-59$ | 16 | 41.3 | 12 | 6.2 | 2 | 7.4 | $<0.001$ |
| $60-64$ | 19 | 49.5 | 25 | 19.2 | 3 | 18.1 | $<0.01$ |
| $65-69$ | 28 | 72.4 | 29 | 34.2 | 3 | 19.4 | $<0.01$ |
| Total | 68 | 46.6 | 80 | 10.7 | 14 | 7.6 |  |
| Age-adjusted |  | 35.4 |  | 10.7 |  | 10.4 |  |

## Definite and possible cases

| $45-49$ | 0 | -- | 1 | 2.0 | 4 | 5.5 | NS |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: | ---: |
| $50-54$ | 7 | 28.5 | 21 | 7.3 | 5 | 9.6 | $<0.01$ |
| $55-59$ | 17 | 43.9 | 18 | 9.3 | 3 | 1.0 | $<0.001$ |
| $60-64$ | 23 | 59.9 | 31 | 23.8 | 3 | 18.1 | $<0.01$ |
| $65-69$ | 33 | 85.3 | 41 | 48.4 | 4 | 25.8 | $<0.01$ |
| Total | 80 | 54.9 | 112 | 15.0 | 19 | 10.3 |  |
| ge-adjusted <br> rate |  | 42.5 |  | 15.0 |  | 13.0 |  |

${ }^{\text {a }}$ In Japan, $85 \%$ of definite and possible stroke cases were seen by the neurologist. Remaining cases diagnosed by neurologist from review of clinic records and occasionally from hospital records.
bIn Hawaii, $62 \%$ of definite and possible stroke cases were seen by the neurologist. Remaining cases diagnosed by neurologist from review of clinic and hospitalization records. The latter were usually available.
${ }^{\text {c }}$ In California, $74 \%$ of definite and possible stroke cases were seen by the neurologist. Remaining cases were classified as definite stroke or no stroke on the basis of the screening test results.
$\mathrm{d}_{\text {Chi }}$ square test with two degrees of freedom.
$\mathrm{e}_{\text {Age-adjusted }}$ by direct method to age structure of Hawaii cohort.

From: "Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: Prevalence of stroke," Kagan, A. et al. In: Cerebrovascular Diseases, Ed. P. Scheinberg, Raven Press, New York, 1976

## TABLE 66

Average Annual Incidence of Definite and Possible Stroke Per 1,000 By Age

| Age | Japan 1972-78 |  |  | Hawaii 1965-73 |  |  | Test ${ }^{\dagger \dagger}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | No. of subjects | No. of cases | Rate ${ }^{\dagger}$ | No. of subjects | No. of cases | Rate |  |
| 45-49 | 54 | 0 | 0.0 | 1825 | 11 | 1.0 | NS |
| 50-54 | 239 | 7 | 7.3 | 2766 | 39 | 2.3 | ** |
| 55-59 | 367 | 11 | 7.5 | 1569 | 21 | 2.2 | *** |
| 60-64 | 357 | 17 | 11.9 | 1306 | 37 | 4.7 | *** |
| 65-69 | 349 | 27 | 19.3 | 429 | 18 | 7.0 | *** |
| Total | 1366 | 62 | 11.3 | 7895 | 126 | 2.7 |  |
| $\begin{aligned} & \text { Age adjusted } \\ & \text { rate }{ }^{\dagger \dagger \dagger} \end{aligned}$ |  |  | 7.4 |  |  | 2.7 | *** |

${ }^{\dagger}$ Annual incidence rate is calculated as follows: Japan: (No. of cases/No. of subjects) 4 (Years follow-up). Hawaii: (No. of cases/No. of subjects) 6/Years follow-up).
$\dagger{ }^{\dagger} \mathrm{X}^{2}$ test of two rates between two cohorts. NS: $\underline{p}>0.10$. *: $\underline{p}<0.05$. **: $\mathrm{p}<0.01 . \quad * * *: \underline{p}<0.001$.
$\dagger \dagger^{\dagger}$ Calculated by the indirect method with Hawaii as standard.

From: "Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: Incidence of stroke in Japan and Hawaii," Takeya, Y. and Popper, J.S. Stroke 15:15-23, 1984

## TABLE 67

Number of Stroke Cases By Subtype and Certainty of Diagnosis .Japan and Hawaii

| Subiype | Japan |  |  | Hawjii |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Total | Defi. nite | Possible | Total | $\begin{aligned} & \text { Defi- } \\ & \text { aite } \end{aligned}$ | Pos. sible |
| Total | 62 | 38 | 24 | 126 | 71 | 33 |
| ICH | 18 | 12 | 6 | 34 | 26 | 8 |
| T-E | 4 | 26 | 18 | 24 | 42 | 32 |
| Unknown | 0 | 0 | 0 | 18 | 3 | 15 |

[^27]
## Average Annual Stroke Incidence Per 1,000 By Age and Subtype -Definite Cases Only

| Age | Total |  |  |  | Intracranial hemorrhage |  |  |  | Thromboembolic stroke |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\frac{\mathrm{Jap}}{\mathrm{No} . \mathrm{of}}$ cases | $\qquad$ <br> Rate | $\frac{\text { Hawa }}{\text { No. of }}$ <br> cases | $\frac{\text { ii }}{\text { Rate }}$ | Jap cases | $\qquad$ <br> Rate | $\frac{\text { Haw }}{\text { No. of }}$ cases | $\frac{e^{\text {Rii }}}{\text { Rate }}$ | Nap cases | $\qquad$ <br> Rate | $\begin{aligned} & \frac{\text { Haw }}{\text { No. of }} \\ & \text { cases } \end{aligned}$ | $\frac{\text { aii }}{\text { Rate }}$ |
| 45-49 | 0 | 0.0 | 6 | 0.55 | 0 | 0.0 | 2 | 0.18 | 0 | 0.0 | 3 | 0.27 |
| 50-54 | 6 | 6.3 | 22 | 1.3 | 4 | 4.2 | 10 | 0.60 | 2 | 2.1 | 11 | 0.66 |
| 55-59 | 7 | 4.8 | 13 | 1.4 | 2 | 1.4 | 3 | 0.32 | 5 | 3.4 | 10 | 1.1 |
| 60-64 | 11 | 7.7 | 21 | 2.7 | 2 | 1.4 | 9 | 1.1 | 9 | 6.3 | 12 | 1.5 |
| 65-69 | 14 | 10.0 | 9 | 3.5 | 4 | 2.9 | 2 | 0.77 | 10 | 7.2 | 6 | 2.3 |
| Total | 38 | 7.0 | 71 | 1.5 | 12 | 2.2 | 26 | 0.55 | 26 | 4.8 | 42 | 0.89 |
| Age adjusted rate |  | 4.7 |  | 1.5 |  | 1.7 |  | 0.55 |  | 3.0 |  | 0.89 |

From: "Epidemiologic studies of coronary heart disease and stroke in Japanese men living Japan, Hawaii, and California: Incidence of stroke in Japan and Hawaii," Takeya, Y and Popper, J.S. Stroke 15:15-23,1984

## TABLE 69

```
Mean Serum Cholesterol Levels At Baseline（1967－1970）For Japanese Men in Japan，Hawaii，and California （from reference 15，Table 3；mg\％）：
```

| AGE | JRPAN | HAWAI I | CALIFORNIA |
| :---: | :---: | :---: | :---: |
| 45－49 | 179.8 | 219.4 | そここ． 4 |
| 50－54 | 1 1日こ． 5 | 219.4 | こモB． |
| 55－59 | 181.5 | E18．7 | だも． 8 |
| 60－64 | 183.2 | 2：6．7 | 2ころ．$\frac{1}{}$ |
| 65－69 | 180.9 | 211．1 | e24．0 |

＊A footnote to the source table notes that the values from Japan were taken from the 1967 cycle，and that diabetes was excluded from this analysis－－therefore other published values may differ slightly

From：＂Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan，Hawaii，and California：Distrib－ ution of biochemical risk factors＂，Nichaman，M．Z．et al． American Journal of Epidemiology 102：491－501， 1975

## TABLE 70

## Case-Fatality Rates in Japanese Men

| -2.0 | $\frac{1967-70}{37.0 \%}$ | $\frac{1971-74}{39.0 \%}$ | $\frac{1975-78}{}$ |
| :--- | ---: | :---: | :---: |
| $56-59 \mathrm{yrs}$ | $36.0 \%$ | $42.0 \%$ | $36.0 \%$ |
| $60-63 \mathrm{yrs}$ | $46.0 \%$ | $45.0 \%$ | $33.0 \%$ |
| $64-67 \mathrm{yrs}$ |  | $45.0 \%$ |  |

From: "Trends in coronary heart disease among men of Japanese ancestry in Hawaii", Reed, D. et al. Journal of Community Health 8:149159, 1983

TABLE 71

Cigarette Smoking Variables Among Chinese-American Men and Women In the California Hypertension Survey, 1979 (from reference 27, table 7.5; comparison data for whites)

|  | $\begin{gathered} \text { Men } \\ 18-49 \end{gathered}$ | $\begin{gathered} \text { Men } \\ 50+ \end{gathered}$ | $\begin{aligned} & \text { Women } \\ & 18-49 \end{aligned}$ | Women 50+ |
| :---: | :---: | :---: | :---: | :---: |
| \%current regular smokers | $\begin{array}{r} 26.6 \\ {[34.6} \end{array}$ | $\begin{aligned} & 35.2 \\ & 30.5 \end{aligned}$ | $\begin{array}{r} 3.6 \\ 36.5 \end{array}$ | $\begin{gathered} 9.7 \\ 25.9 \text { ] } \end{gathered}$ |
| \%current or former smokers | $\begin{array}{r} 42.0 \\ {[58.0} \end{array}$ | $\begin{aligned} & 69.8 \\ & 72.6 \end{aligned}$ | $\begin{array}{r} 7.7 \\ 52.2 \end{array}$ | $\begin{aligned} & 19.2 \\ & 48.7] \end{aligned}$ |
| average cigarettes <br> per day among <br> current smokers | $\begin{array}{r} 12.5 \\ {[26.5} \end{array}$ | $\begin{aligned} & 16.4 \\ & 28.3 \end{aligned}$ | $\begin{aligned} & 12.0 \\ & 23.4 \end{aligned}$ | $\begin{aligned} & 17.1 \\ & 23.2] \end{aligned}$ |
| \%of current smokers who would like to quit | $\begin{array}{r} 54.3 \\ {[71.0} \end{array}$ | $\begin{aligned} & 59.0 \\ & 59.0 \end{aligned}$ | $\begin{aligned} & 48.0 \\ & 63.8 \end{aligned}$ | $\begin{aligned} & 66.5 \\ & 60.8 \text { ] } \end{aligned}$ |

From: "Ischemic heart disease risk factors in Asian Americans", Kumanyika, S.K. and Savage, D.D. Paper commissioned by the Task Force on Black and Minority Health, 1984-1985

TABLE 72

Cigarette Smoking Variables Among Filipino-American Men and Women In the California Hypertension Survey, 1979 (from reference 27, table 7.5; comparison data for whites)

|  | $\begin{gathered} \text { men } \\ 18-49 \end{gathered}$ | $\begin{aligned} & \text { Men } \\ & 50+ \end{aligned}$ | Wamen $18-49$ | Wamen 50+ |
| :---: | :---: | :---: | :---: | :---: |
| xcurrerit reguiar smoners | $\begin{array}{r} 30.7 \\ {[\quad 34.6} \end{array}$ | $\begin{aligned} & 21.2 \\ & 30.5 \end{aligned}$ | $\begin{aligned} & 12.4 \\ & 36.5 \end{aligned}$ | $\begin{aligned} & 16.1 \\ & 25.9 \mathrm{I} \end{aligned}$ |
| Xcurrent or former smokers | $\begin{array}{r} 58.3 \\ =58.0 \end{array}$ | $\begin{aligned} & 67.2 \\ & 72.6 \end{aligned}$ | $\begin{aligned} & 3 \text { 3e. } 9 \\ & 5 \Xi .2 \end{aligned}$ | $\begin{aligned} & 25.9 \\ & 48.72 \end{aligned}$ |
| average cigarettes per day among current smokers | $\begin{array}{r} 18.2 \\ \mathrm{E} . \mathrm{E} . \mathrm{S} \end{array}$ | $\begin{aligned} & 17.2 \\ & 28.3 \end{aligned}$ | $\begin{aligned} & 15.0 \\ & 25.4 \end{aligned}$ | $\begin{array}{r} 1.1 \\ 23.2 j \end{array}$ |
| Kof current smokers who woule like te quit | $\begin{aligned} & 75.6 \\ & \mathrm{c} \quad 71.0 \end{aligned}$ | $\begin{aligned} & 90.8 * \\ & 59.0 \end{aligned}$ | $\begin{aligned} & 67.6 * \\ & 63.8 \end{aligned}$ | $\begin{aligned} & 13.2 * \\ & 60.8 \mathrm{j} \end{aligned}$ |

* Based on fewer than 20 cases

From: "Ischemic heart disease risk factors in Asian Americans", Kumanyika, S.R. and Savage, D.D. Paper commissioned by the Task Force on Black and Minority Health, 1984-1985

## TABLES

NATIVE AMERICANS

Tables 80-83

## TABLE 80

Leading Causes of Death Among the U.S., All Races, 1979, and Comparable Data For Indians and Alaska Natives, 1978-1980

## Percent Distribution

```
Leading Causes
U.S. All Races
```

Olseases of the heart

```
Olseases of the heart
Malignant neoplasms
Malignant neoplasms
cerebrovascular diseases
cerebrovascular diseases
Accidents
Accidents
COPO.
COPO.
pneumonia and inftuenza
pneumonia and inftuenza
glabetes mellitus
glabetes mellitus
glabetes mellitus
glabetes mellitus
    and cirrhosis.e
    and cirrhosis.e
Atherosclerosis
Atherosclerosis
sufcide
sufcide
all other
all other
all causes
```

```
all causes
```

```
U.S.
All Races
inaians and
38.3
21.1
8.9
5.5
2.6 2.4 Alaska Natives
    1.7
    1.6
        1.5
        1.4
    15.1
    100.0
20.8
10.1
4.8
19.5
0.9
3.8
2.9
6.0
0.7
2.6
28.04
100.0
* Chronic obstructive pulmonary disease and allied conditions :h:C Cirrhosis of the liver, 1978
\# Among Indians and Alaska Natives, "all other" includes 3.3\% homicide, \(2.6 \%\) suicide, and \(2.7 \%\) deaths attributed to "certain causes of mortality in early infancy (1978)"

From: "Ischemic heart disease risk factors among American Indians and Alaska Natives", Kumanyika, S.K. and Savage, D.D. Paper commissioned by the Task Force on Black and Minority Health, 1984-85

\section*{TABLE 81}

Comparison of Mortality Rates For Indians and Alaska Natives With Those of the General Population and Other U.S. Non-White Populations In Two Time Periods For Selected Causes
\begin{tabular}{|c|c|c|c|c|}
\hline \multicolumn{2}{|l|}{cause of death CLASSIFICATION} & RATE & \begin{tabular}{l}
batio to \\
U.S. All Races
\end{tabular} & RATIO TO U.S. other non-whites \\
\hline Infant & 1955 & 62.7 . & 2.4 & 1.5 \\
\hline Mortality & 1979 & 14.6* & 1.1 & 0.7 \\
\hline Tuber- & 1955 & 57.9: & 6.9 & 2.4 \\
\hline culosis & 1980 & \(3.6 \pm\) & 6.0 & 1.5 \\
\hline \multirow[t]{2}{*}{Gastrointestinal disease} & & & & \\
\hline & \[
\begin{aligned}
& 1955 \\
& 1980
\end{aligned}
\] & \[
15.4 .
\] & 1.3 & 2.3
1.3 \\
\hline \multirow[t]{2}{*}{Accidents} & 1955 & 184.04\% & 3.3 & 2.6 \\
\hline & 1980 & 107.30 F & 2.5 & 2.1 \\
\hline \multirow[t]{2}{*}{Alconolisn} & 1969 & 56.6! & 7.4 & not given \\
\hline & 1980 & 41.3! & 5.5 & \\
\hline
\end{tabular}
* Deaths per 1,000 live births; from reference 2, table 3.3
\# Age-adjusted deaths per 100,000 population; from ref 2, table 4.11 :!: " " " " " " " " " " " " " " " " " " , table 4.13
非 ", "" ", " " " " " " " " " " " " " " " " " " " " " " " " " "
! " " " " " " " " " " " " " " " " " " , table 4.10

From: "Ischemic heart disease risk factors among American Indians and Alaska Natives", Kumanyika, S.K. and Savage, D.D. Paper commissioned by the Task Force on Black and Minority Health, 1984-85

\title{
TABLE 82
}

Mortality, U.S., 1979-1981: Excess Deaths From Heart Disease
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline \multirow[b]{3}{*}{Age} & \multicolumn{12}{|c|}{\[
\begin{gathered}
390 \text { to } 398402404 \text { to } 429 / \text { Hesrt Disesse } \\
\text { Race }=\text { White }
\end{gathered}
\]} \\
\hline & \multicolumn{6}{|c|}{Male} & \multicolumn{6}{|c|}{Female} \\
\hline & Deaths & \[
\begin{gathered}
\text { Rste/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul & Deaths & \[
\begin{gathered}
\text { Rate/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul \\
\hline Under 1 & 303 & 21.71 & 0 . & 0. & * & * & 219 & 16.54 & 0 . & 0 . & * & * \\
\hline 1-4 & 111 & 2.19 & 0. & 0. & * & * & 103 & 2.14 & 0. & 0. & * & * \\
\hline 5-9 & 55 & 0.82 & 0. & 0. & * & * & 53 & 0.83 & 0. & 0. & * & * \\
\hline 10-14 & 77 & 1.03 & 0. & 0. & * & * & 54 & 0.77 & 0. & 0. & * & * \\
\hline 15-19 & 188 & 2.17 & 0. & 0. & * & * & 111 & 1.34 & 0. & 0. & * & * \\
\hline 20-24 & 317 & 3.65 & 0. & 0. & * & * & 176 & 2.05 & 0. & 0. & * & * \\
\hline 25-29 & 498 & 6.22 & 0. & 0. & * & * & 240 & 3.01 & 0. & 0. & * & * \\
\hline 30-34 & 1021 & 13.99 & 0. & 0. & * & * & 400 & 5.45 & 0. & 0. & * & * \\
\hline 35-39 & 2258 & 38.72 & 0. & 0. & * & * & 652 & 10.99 & 0. & 0. & * & * \\
\hline 40-44 & 4615 & 95.16 & 0. & 0. & * & * & 1225 & 24.62 & 0. & 0. & * & * \\
\hline 45-49 & 9218 & 198.73 & 0. & 0. & * & * & 2385 & 49.49 & 0. & 0. & * & * \\
\hline 50-54 & 17395 & 353.70 & 0. & 0. & * & * & 4958 & 94.63 & 0. & 0. & * & * \\
\hline 55-59 & 28276 & 582.67 & 0. & 0. & * & * & 9444 & 175.38 & 0. & 0. & * & * \\
\hline 60-64 & 38527 & 923.22 & 0. & 0. & * & * & 15993 & 333.01 & 0. & 0. & * & * \\
\hline 65-69 & 49198 & 1413.08 & 0. & 0. & * & * & 25260 & 583.28 & 0. & 0. & * & * \\
\hline 70-74 & 55227 & 2163.78 & 0. & 0. & * & * & 37014 & 1044.69 & 0. & 0. & * & * \\
\hline 75-79 & 53635 & 3249.85 & 0. & 0. & * & * & 49236 & 1851.04 & 0. & 0. & * & * \\
\hline 80-84 & 46245 & 5008.62 & 0. & 0. & * & * & 59807 & 3394.17 & 0. & 0. & * & * \\
\hline 85-P1us & 54353 & 8846.47 & 0. & 0. & * & * & 105794 & 7395.32 & 0. & 0. & * & * \\
\hline All Ages & 361516 & 394.30 & 0. & 0. & * & * & 313124 & 323.86 & 0. & 0. & * & * \\
\hline
\end{tabular}
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline & \multicolumn{6}{|c|}{Male} & \multicolumn{6}{|l|}{Black Female} \\
\hline Age & Desths & \[
\begin{gathered}
\text { Rate/ } \\
100,000
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \% Cumul & Deaths & \[
\begin{gathered}
\text { Rate/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \% Cumul \\
\hline Under 1 & 111 & 41.44 & 53. & 53. & 1.59 & 1.59 & 99 & 37.64 & 56. & 56. & 1.95 & 1.95 \\
\hline 1-4 & 40 & 5.10 & 28. & 81. & 7.05 & 2.17 & 40 & 4.20 & 19. & 75. & 5.96 & 2.36 \\
\hline 5-9 & 12 & 0.96 & 2. & 82. & 0.95 & 2.11 & 16 & 1.27 & 5. & 81. & 5.32 & 2.45 \\
\hline 10-14 & 23 & 1.71 & 9. & 91. & 10.06 & 2.30 & 22 & 1.68 & 12. & 93. & 17.30 & 2.76 \\
\hline 15-19 & 71 & 4.75 & 38. & 130. & * & 3.26 & 47 & 3.14 & 27. & 120. & * & 3.56 \\
\hline 20-24 & 127 & 9.79 & 80. & 210. & 7.48 & 4.15 & 81 & 5.71 & 52. & 172. & 12.00 & 4.53 \\
\hline 25-29 & 216 & 19.92 & 149. & 358. & 7.30 & 5.06 & 140 & 11.29 & 102. & 274. & 12.57 & 5.95 \\
\hline 30-34 & 357 & 40.99 & 235. & 593. & 9.72 & 6.24 & 202 & 19.88 & 147. & 421. & 14.89 & 7.53 \\
\hline 35-39 & 607 & 91.59 & 350. & 944. & 14.31 & 7.90 & 319 & 40.11 & 232. & 653. & 20.12 & 9.67 \\
\hline 40-44 & 1041 & 183.75 & 502. & 1446. & 18.09 & 9.82 & 570 & 83.27 & 401. & 1054. & 27.43 & 12.84 \\
\hline 45-49 & 1717 & 333.15 & 693. & 2138. & 20.33 & 11.79 & 951 & 151.57 & 641. & 1695. & 32.14 & 16.62 \\
\hline 50-54 & 2706 & 536.41 & 922. & 3060. & 21.30 & 13.62 & 1577 & 252.56 & 986. & 2681. & 36.44 & 20.77 \\
\hline 55-59 & 3866 & 828.71 & 1148. & 4208. & 23.21 & 15.35 & 2326 & 407.87 & 1326. & 4007. & 39.29 & 24.61 \\
\hline 60-64 & 4502 & 1169.11 & 947. & 5155. & 21.57 & 16.21 & 3206 & 660.03 & 1589. & 5596. & 43.78 & 28.10 \\
\hline 65-69 & 5166 & 1557.31 & 478. & 5633. & 13.58 & 15.95 & 4236 & 951.40 & 1639. & 7235. & 44.17 & 30.63 \\
\hline 70-74 & 5154 & 2199.75 & 84. & 5717. & 3.83 & 15.24 & 4882 & 1482.86 & 1443. & 8677. & 40.43 & 31.91 \\
\hline 75-79 & 4540 & 2972.08 & 0. & 5717. & 0.00 & 15.02 & 5095 & 2171.52 & 752. & 9429. & 35.42 & 32.17 \\
\hline 80-84 & 3345 & 4463.36 & 0. & 5717. & * & 15.02 & 4304 & 3442.27 & 60. & 9489. & 7.80 & 31.54 \\
\hline 85-Plus & 3434 & 6482.78 & 0. & 5717. & * & 15.02 & 5888 & 5557.13 & 0. & 9489. & * & 31.54 \\
\hline All Agea & 37043 & 295.89 & 5717. & -- & 15.02 & * & 34003 & 243.30 & 9489. & -- & 31.54 & * \\
\hline
\end{tabular}

\footnotetext{
*Percent values are not given when the base of calculation (the excess deaths from all causeg for a particular sge, sex, and racial group) is equal to zero.
From: Mortality Rates, Excess Deaths. National Center for Hesith Statistics' Death Certificate Data Tapes for 1979 , 1980 , and 1981. (Tables supplied by the DHHS Task Force on Black and Minority Health.)
}

TABLE 82 (Continued)
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline \multirow[b]{3}{*}{Age} & \multicolumn{12}{|c|}{\[
\begin{gathered}
390 \text { to } 398402404 \text { to } 429 / \text { Heart Disease } \\
\text { Rsce }=\text { Indian }
\end{gathered}
\]} \\
\hline & \multicolumn{6}{|r|}{Male} & \multicolumn{6}{|c|}{Female} \\
\hline & Deaths & \[
\begin{gathered}
\text { Rate/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul & Deaths & \[
\begin{gathered}
\text { Rate/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul \\
\hline Under 1 & 5 & 32.28 & 2. & 2. & 3.57 & 3.57 & 4 & 24.48 & 1. & 1. & 2.45 & 2.45 \\
\hline 1-4 & 2 & 2.81 & 0. & 2. & 1.76 & 3.02 & 3 & 5.25 & 2. & 3. & 7.94 & 4.09 \\
\hline 5-9 & 0 & 0.00 & 0. & 2. & 0.00 & 2.73 & 1 & 0.92 & 0. & 3. & 1.49 & 3.94 \\
\hline 10-14 & 0 & 0.42 & 0. & 2. & 0.00 & 2.41 & 1 & 1.73 & 1. & 4. & 33.83 & 4.74 \\
\hline 15-19 & 2 & 1.93 & 0. & 2. & 0.00 & 1.31 & 2 & 2.78 & 1. & 5. & 5.67 & 4.94 \\
\hline 20-24 & 6 & 8.02 & 3. & 5. & 2.28 & 1.76 & 3 & 3.59 & 1. & 6. & 2.63 & 4.25 \\
\hline 25-29 & 8 & 12.45 & 4. & 9. & 3.00 & 2.13 & 3 & 4.73 & 1. & 7. & 1.98 & 3.63 \\
\hline 30-34 & 9 & 17.17 & 2. & 11. & 1.52 & 2.00 & 6 & 10.34 & 3. & 1. & 4.83 & 3.89 \\
\hline 35-39 & 28 & 68.89 & 12. & 23. & 11.40 & 3.57 & 6 & 13.16 & 0. & 11. & 1.97 & 3.59 \\
\hline 40-44 & 38 & 83.93 & 0. & 23. & 0.00 & 3.10 & 12 & 33.70 & 0. & 14. & 6.10 & 3.96 \\
\hline 45-49 & 45 & 162.30 & 0. & 23. & 0.00 & 2.88 & 14 & 47.53 & 0. & 14. & 0.00 & 3.61 \\
\hline 50-54 & 63 & 253.55 & 0. & 23. & 0.00 & 2.81 & 23 & 87.67 & 0. & 14. & 0.00 & 3.41 \\
\hline 55-59 & 89 & 412.96 & 0. & 23. & * & 2.81 & 33 & 142.29 & 0. & 14. & * & 3.41 \\
\hline 60-64 & 101 & 632.94 & 0. & 23. & * & 2.81 & 49 & 275.47 & 0. & 14. & * & 3.41 \\
\hline 65-69 & 113 & 879.58 & 0. & 23. & * & 2.81 & 75 & 482.87 & 0. & 14. & * & 3.41 \\
\hline 70-74 & 105 & 1182.94 & 0. & 23. & * & 2.81 & 79 & 712.24 & 0. & 14. & * & 3.41 \\
\hline 75-79 & 100 & 1645.08 & 0. & 23. & * & 2.81 & 75 & 974.63 & 0. & 14. & * & 3.41 \\
\hline 80-84 & 72 & 2506.96 & 0. & 23. & * & 2.81 & 80 & 1898.88 & 0. & 14. & * & 3.41 \\
\hline 85-P1us & 90 & 3894.42 & 0. & 23. & * & 2.81 & 113 & 3164.79 & 0. & 14. & * & 3.41 \\
\hline All Ages & 867 & 123.46 & 23. & -- & 2.81 & * & 581 & 80.95 & 14. & -- & 3.41 & * \\
\hline & \multicolumn{12}{|c|}{Male Race \(=\) Asisu} \\
\hline Age & Desths & \[
\begin{gathered}
\text { Rate/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul & Desths & \[
\begin{gathered}
\text { Rste/ } \\
100,000 \\
\hline
\end{gathered}
\] & Ex Dth & Cumul & \%Ex Dth & \%Cumul \\
\hline Under 1 & 9 & 27.77 & 2. & 2. & * & * & 5 & 17.55 & 0. & 0. & * & * \\
\hline 1-4 & 4 & 3.13 & 1. & 3. & * & * & 3 & 2.32 & 0. & 1. & * & * \\
\hline 5-9 & 1 & 0.43 & 0. & 3. & * & * & 1 & 0.90 & 0. & 1. & * & * \\
\hline 10-14 & 2 & 1.16 & 0. & 3. & * & * & 2 & 1.47 & 1. & 2. & * & * \\
\hline 15-19 & 4 & 2.68 & 1. & 4. & * & * & 1 & 0.96 & 0. & 2. & * & * \\
\hline 20-24 & 8 & 4.88 & 2. & 6. & * & * & 4 & 2.25 & 0. & 2. & * & * \\
\hline 25-29 & 7 & 4.31 & 0. & 6. & * & * & 7 & 3.52 & 1. & 3. & * & * \\
\hline 30-34 & 18 & 10.44 & 0. & 6. & * & * & 9 & 4.29 & 0. & 3. & * & * \\
\hline 35-39 & 24 & 18.24 & 0. & 6. & * & * & 9 & 5.97 & 0. & 3. & * & * \\
\hline 40-44 & 45 & 42.03 & 0. & 6. & * & * & 15 & 13.29 & 0. & 3. & * & * \\
\hline 45-49 & 66 & 80.14 & 0. & 6. & * & * & 22 & 22.36 & 0. & 3. & * & * \\
\hline 50-54 & 115 & 185.18 & 0. & 6. & * & * & 32 & 36.41 & 0. & 3. & * & * \\
\hline 55-59 & 168 & 275.02 & 0. & 6. & * & * & 53 & 77.45 & 0. & 3. & * & * \\
\hline 60-64 & 178 & 394.49 & 0. & 6. & * & * & 79 & 150.66 & 0. & 3. & * & * \\
\hline 65-69 & 240 & 636.59 & 0 . & 6. & * & * & 100 & 245.84 & 0. & 3. & * & * \\
\hline 70-74 & 345 & 1114.02 & 0. & 6. & * & * & 124 & 456.69 & 0. & 3. & * & * \\
\hline 75-79 & 349 & 1780.79 & 0. & 6. & * & * & 172 & 882.41 & 0. & 3. & * & * \\
\hline 80-84 & 256 & 2842.95 & 0. & 6. & * & * & 172 & 1427.98 & 0. & 3. & * & * \\
\hline 85-P1us & 293 & 5631.45 & 0. & 6. & * & * & 302 & 3485.46 & 0. & 3. & * & * \\
\hline All Ages & 2140 & 126.36 & 6. & -- & * & * & 1111 & 61.50 & 3. & -- & * & * \\
\hline
\end{tabular}

\section*{TABLE 83}
```

Percent Change In Age-Adjusted Mortality (Rate Per 100,000 Population)
Between }1970\mathrm{ and 1975 For Indians and Alaska Natives
And the U.S., All Races

```
\begin{tabular}{|c|c|c|c|}
\hline Cause of Death & Indians and Alasta Natives & \begin{tabular}{l}
U.S. \\
All Races
\end{tabular} & ratio* \\
\hline Disease of the Heart & -12.7 & -13.1 & 0.7 \\
\hline Cerebrovascular & -20.9 & -17.8 & 0.7 \\
\hline Arterioscterosis & -14.4 & -21.4 & 1.5 \\
\hline Hypertens ion & - 5.9 & -34.5 & 0.8 \\
\hline Diabetes Mellitus & -12.2 & -17.7 & 2.1 \\
\hline Homictae & -19.4 & +15.4 & 2.5 \\
\hline Suicide Cirinasts of the liver & 195.3
+4.9 & 16.8
-6.1 & 2.1 \\
\hline
\end{tabular}
\(\therefore 1975\) ratio of rate for Indians and Alaska Natives to rate for U.S., all races

\footnotetext{
From: "Ischemic heart disease risk factors among American Indians and Alaska Natives", Kumanyika, S.K. and Savage, D.D. Paper commissioned by the Task Force on Black and Minority Health, 1984-85
}

\section*{RECOMMENDATIONS MADE BY THE SUBCOMMITTEE ON CARDIOVASCULAR AND CEREBROVASCULAR DISEASES IN BLACK AND MINORITY HEALTH}

\section*{A. Research Issues}

\section*{1) Research in Epidemiology and Etiology}

Minority ethnic American groups, whether of primarily Black, Hispanic, Asian, or Native American descent, share many health problems in common as well as displaying certain unique disease characteristics. These differentials may be unique to a certain age-group, or to gender, or to the geographic location of the minority group. The etiology and epidemiology of illness in such groups warrant careful investigation, not only in cases where a clear disease burden is evident but also for diseases in which a minority subgroup displays better health than the comparable majority population.

CVD-specific research recommendations include:
(a) Large-scale, population-based, prospective studies of coronary heart disease (CHD) - similar to the Framingham Studies - and/or community-based studies are needed for each of the minority populations. Of the many Hispanic subgroups, Puerto Ricans and Cubans particularly warrant such research. A key component of this research would be the validation in minorities of the major established and/or suspected biological risk factors for CHD that have been identified for the white American population. These risk factors include hypertension, obesity, hypercholesterolemia, inadequate physical activity, cigarette smoking, diabetes mellitus, apolipoprotein imbalance, and echocardiographic and ECG-LVH. Another key element of such research would be the surveillance of the offspring of indexed cases so as to provide crucial information on trends for risk and for disease as well as elucidating familial contribution to CHD incidence and process. Further studies on potential differences in sudden death rates between Blacks and whites, by age and gender, are needed.
(b) The investigation of the impact of other diseases, such as influenza, on CHD mortality rates in minority groups is needed.
(c) The effect of changes in the cardiovascular disease classification system on assessing trends in cause-specific mortality rates in minorities needs to be better documented.
(d) Research on why excess deaths due to cardiovascular disease (CVD) in Native American men occur in the 35-39 age-group, but not in.older men, is needed. The association of CHD with age is less in Native Americans than in white Americans.

There is a need to better understand risk status, risk profiles, and trends in risk patterns for the major health priority areas, for each minority group. In large part such research can be accomplished by well-designed, epidemiologic studies and/or by case-control studies, as mentioned in 1). Certain minority subsets have a more favorable health status for certain disease or have reduced all-cause mortality. For example, in every age-group, Chinese, Japanese, and Filipino American have lower all-cause mortality rates than do white, Black, and Native Americans. Not only risk factors but also environmental factors should be examined for their contribution to increased prevalence of diabetes, infectious diseases, coronary heart disease, cancer, and for the high prenatal mortality found during the past four decades in one or more of the four minorities that are the subject of this report.

CVD-specific research recommendations include:
(a) Biological risk factors for CHD, identified and/or suspected for whites, need to be validated and quantified for each minority.
(b) Study of the determinants of nicotine and alcohol use behaviors, cessation, and cessation maintenance in minorities is needed.
(c) Investigation of whether and to what extent high HDL-levels in Black men may confer benefit with regard to CHD outcomes is recommended.
(d) A more complete risk profile analysis of the Black participants in the Hypertension Detection and Follow-Up Program, and the Multiple Risk Factor Intervention Trial should be undertaken.
(e) Case-control studies in Hispanics are suggested to clarify the hypothesised relationships between amount of Indian admixture and "tolerance" for noninsulin-dependent diabetes mellitus and of percentage of Black admixture as a risk factor for hypertension.
(f) Better assessment of the impact of hypertension on the morbidity/mortality gap in Blacks is needed.
(g) Tribal variation in diabetes mellitus, a risk factor for CHD, among middle-aged and older Native Americans needs considerably more attention and research.
(h) The value of 24 -hour monitoring of blood pressure, the most sensitive known measure of hypertension, in predicting subsequent CHD risk in minority groups needs to be studied.
3) Research on Diagnosis and Treatment

Research is needed to better understand the dynamics of medical care available to Black, Hispanic, Asian, and Native Americans. Appropriate diagnosis, treatment, and follow-up predispose for a favorable outcome.

CVD-specific research recommendations include:
(a) Investigation is needed on how specific patterns of risk factors in minorities influence treatment decisions such as whether or not to perform angioplasty or coronary bypass surgery.
(b) Studies are needed of beliefs, awareness status, and pre-hospital behavior which might delay appropriate diagnosis and treatment for individuals with symptoms of CHD in minority communities.
(c) Research is needed on whether the Rose Questionnaire diagnosis of angina pectoris as an indicator of CHD is less specific for Black women than for white.
(d) Effective stress-reduction and behavior modification strategies for treatment of CHD and risk factors need to be developed and validated in minority populations. Successful techniques should be taught to the deliverers as well as to the receivers of health care.
(e) Do Blacks receiving dialysis for hypertension-related end-stage renal disease (ESRD) have lower death rates, after adjustment for age, than whites do? If so, why?
(f) Compliance/noncompliance to antihypertensive medication regimens needs to be studied. Why do Filipino women have poorer blood pressure control than Filipino men? This is in contrast to other ethnic minority groups in the United States in which women generally have better blood pressure control than men.
(g) The long-term efficacy and safety of antihypertensive medications prescribed to minorities (particularly Blacks) need to be examined. Do the metabolic, hemodynamic and side effects of treatments and their impact on CHD differ among minorities and whites?

\section*{4) Research on Nutritional Factors}

Research on nutritional patterns, status, needs, and health consequences must specifically address and define differences between minority and majority populations. Profiles of physiological factors, cultural food patterns, and dietary behaviors and intake all need to be studied for each ethnic/racial group by gender, by age, and by socioeconomic status. In addition, nutrition's important role in effecting positive change in minority health status needs
elucidation. Such research would be additional to and distinct from that which investigates nutrition's role as a component of a particular socioeconomic condition.
CVD-specif ic research recommendations include:
(a) Research to determine the relationship of obesity in Black females during and after adolescence, to high-density lipoprotein and low-density lipoprotein serum levels is needed. Development of effective weight-control programs is recommended.
(b) Investigation of the links between dietary potassium, sodium, calcium and, possibly, other dietary elements and hypertension in Blacks and other minorities is needed.
(c) Detailed studies of total dietary cholesterol, of serum cholesterol and triglyceride fractions, as well as dietary risk reduction information, interventions, and behaviors, are recommended for Puerto Rican, Cuban, and other Hispanic populations.
(d) The effect of diet on the development of diabetes mellitus in minority populations needs further study. In particular, the recent interest in the role of upper body adiposity as a risk factor for diabetes, itself a risk factor for CHD, should be pursued more extensively and tested in all Hispanic groups, as well as in Black, Asian, and Native Americans.

\section*{5) Research on Socioeconomic Status and Acculturation}

Sociocultural factors play in integral role in health status, in illness development, and in the treatment process. As new waves of minority immigrants settle in the United States, they bring with them the health/disease profile prevalent in their socioeconomic group from their country of origin. Their process of acculturation as new Americans most likely occurs at differing rates, depending on their past history and new allegiances, within their different minority subgroups. They may trade poor health habits for good, or vice versa. Many factors such as physiological, socioeconomic, behavioral, familial, and racial factors as well as cultural affiliations interact in this acculturation process. Multi-discipline research is needed to investigate this with the goal of maintaining good health habits and retarding the acquisition of unhealthy habits.

CVD-specific research recommendations include:
(a) Investigation of socioeconomic status (SES) as a risk factor for CHD, hypertension, stroke, and hypertension-related end-stage renal disease in all minority groups is needed. What is special about the status of Asian Americans that might confer some degree of protection with regard to CHD mortality on them, despite their having an apparently moderately high-risk profile?
(b) Further research on the associations of social mobility and social status with CHD risk factors in minorities is needed.
(c) More developmental work is needed in Native Americans to produce a valid and reliable measure of acculturation and of sociocultural indices of shifts in beliefs, values, and behavior patterns which might increase CHD risk.
(d) A research hypothesis worthy of investigation is that both low acculturation and high acculturation are associated with increased mortality risk in different Asian groups. Low acculturation may confer greater risk for overall mortality perhaps due to high rates of infectious diseases and high perinatal mortality. High acculturation may increase the mortality risk from diseases associated with more developed, westernized societies, such as coronary heart disease.
6) Research on Health Care Delivery

Primary medical care research is needed to investigate:
-how coordination of the "traditional" health care delivery process can optimize patient outcomes
-determinants of physician behavior with regard to patient interaction, follow-up, and the adoption of innovations (particularly for prevention and early detection)
-health care provider attributes such as physician's ethnicity or race which could affect health service utilization by minority people, whether negatively or positively
-the intersection of folk (or "nontraditional") medicine with the "traditional" health care system
-the extent to which and means by which the poor and the near-poor gain access to health facilities, and how they cope with any lack of accessibility
-the involvement of the community in facilitating health care access and delivery to minority people.

Multi-center, multi-disciplinary case-control studies are recommended for such research.

CVD-specific research recommendations include:
(a) The need to monitor CHD events that occur in the community, such as sudden death; hospital admissions and discharges of patients
diagnosed to have CHD; and emergency room visits for chest pains and related complaints.
(b) The relative effectiveness of a model such as the Trilateral High Blood Pressure, Detection, and Control Project(a) (a collaborative project by voluntary organizations in the private and public sectors, which produced a manual for instructing community organizers and volunteers on techniques and planning for setting up community-wide, community-run, blood pressure control programs); and model programs such as the State of Georgia's Statewide Antihypertensive Drug Distribution System for Indigent Hypertensives(b) (whereby as many as 32,000 state residents with hypertension, and eligible for free medical care or Medicaid, can receive low-cost antihypertensive medication) need to be evaluated in different minority communities. Successful models for community action to promote cardiovascular health should be provided to minority communities.

\section*{7) Research on Disease Prevention/Health Promotion}

There is an urgent need for rigorous prevention-oriented and policy-relevant research, including mental health research, commensurate with the levels of need and representation of minority populations within this country. For example, certain research shows there are serious and pervasive adjustment problems - economically, socioculturally and psychologically - affecting large segments of the Indochinese refugee population, especially the more recently arrived groups. Research to determine the components of effective disease prevention and health promotion activities targeted toward health education in minority families is needed. Methods which build on the strengths of these families and their communities, both urban and rural, especially social support characteristics should be used.

CVD-specific research recommendations include:
(a) Determination of effective strategies for cardiovascular health education among specific minority groups and how to facilitate the adoption of specific interventions for cardiovascular risk factors, especially among high-risk subgroups. The models of the National High Blood Pressure Education Program can be adopted and modified for other risk factors for a variety of communities. Educational and therapuetic interventions successfully developed for relatively homogeneous groups in a variety of studies should be modified for use with high-risk minority groups. Care must be taken to consider the different cultural values and attitudes towards CHD and risk factors for CHD, such as obesity, chest pain, and particular health behaviors.
(a) The American Red Cross, Publications, 17 and D St NW, Washington, DC
(b) Georgia Dept Human Resources, 878 Peachtree St.NE, Atlanta, GA 30309
8) Research on Health Policy

Research is needed on the effects of subtle changes in Federal policy on the appropriateness of health care received by minority populations. For example, research is required to understand the impact of the following on provider-patient interactions and on care received:
-small changes in reimbursement may lead to reduction in certain services that impact disproportionately in minority populations, or upon high-risk subsets
-policy changes in direct service programs, such as the Indian Health Service, Community Health Centers, Migrant Health Centers, may lead to changes in the management, organization, and delivery of services which then affect continuity and coordination of the care offered
-changes in manpower development policies may affect the availability of health care personnel to minority communities.

\section*{B. Information and Education}
1) Minority health information dissemination should be continued, but with special emphasis on the health needs of each minority group, by age and gender, and with attention given to the most effective approach for that minority subgroup.
2) Studies that assess the need for minority patient education in specific settings, e.g. the hospital emergency room or outpatient clinic, the church, or through home-visits by trained counselors are needed and consequent, appropriate interventions need to be designed and used.
3) The publication and updating of a list of DHHS health promotion and disease prevention materials, including patient education materials, especially directed toward specific minority groups, would be of value to practicing physicians and other health care providers.
4) Targeted health education programs should be developed for specific minorities with consideration given to techniques which will lead to community-wide activation rather than to activation of the individual.
5) Techniques are needed that will encourage earlier diagnosis, full use of all diagnostic procedures, and earlier treatment interventions so that Blacks and other minorities enter the delivery system well before an advanced disease state has developed.
6) There is enough general, if not specific, evidence to justify a recommendation for health promotion interventions directed toward minority groups that would reinforce a diet to lower blood cholesterol, to reduce or eliminate cigarette use, to moderate and to maintain normal body weight and blood pressure.
7) Cardiovascular risk factor educational materials that will facilitate information exchange between the primary care professional and the patient should be developed.
8) Targeted smoking prevention programs for minorities are needed.
9) Continued efforts at education, prevention, treatment and control of the hypertension-related diseases, e.g. stroke and end-stage renal disease, especially in Blacks, are needed.

\section*{C. Access \& Utilization}
1) In addition to genetic, environmental, and behavioral factors, appropriate medical care is a major determinant of morbidity and mortality due to cardiovascular disease. Under optimal medical care conditions, for example, a patient with essential hypertension can achieve blood pressure control and reduce the risk of cardiovascular sequelae. However, with variations in physician behavior and patient care-seeking behavior, optimal medical care circumstances are difficult to achieve for large population groups, and are equally if not more difficult to achieve for most minority populations. Simultaneous attention to all the elements of interaction is necessary.
2) Continue to foster adequate access to care for minorities, with special attention given to unique medical care usage patterns and any financial barriers.
3) Studies of beliefs, awareness, and pre-hospital behavior, which might delay appropriate diagnosis and treatment for individuals with symptoms of coronary heart disease in the minority communities, are needed.
4) Though there is increasing awareness, interest, and sophistication among physicians concerning many aspects of blood pressure (BP) control, programs that attempt to enhance physicians' ability to ensure optimal follow-up of hypertensive patients and to monitor the state of BP control continue to be needed.
5) Continued efforts at prevention, treatment, and control of the hypertension-related diseases, e.g. stroke and end-stage renal disease are needed, especially in the Black population.
D. Capacity Building in Non-Federal Sector
1) The detailing of Federal workers to minority institutions for short-term assignment is recommended.
2) The opportunity for minority non-Federal workers to come to Federal facilities for training \& experience should be increased.
3) The establishment in the Federal sector of improved liaison and information dissemination programs designed not only to respond to requests from non-Federal minority groups but also to actively involve volunteers from those groups as part of an intentional network-making effort.

\section*{E. Financing}
1) Careful and serious consideration should be given to ways in which health care deliverers could be reimbursed for providing care in preventing disease in minorities.

\section*{F. Health Professions' Development}
1) The detailing of Federal workers to minority institutions for short-term assignment is recommended.
2) The opportunity for minority non-Federal workers to come to Federal facilities for training \& experience should be increased.
3) Development of innovative mechanisms to attract minorities into the health care field \& into health research needs to be undertaken with direct and continuing input from leaders in the minority health professions.
4) Special efforts need be made to continue to aid minority researchers and those in minority research settings to be competitive in seeking research funding.
5) The recommendations of this Task Force need to be presented to numerous specific health professional organizations \& health professional schools.

\section*{G. Leadership}
1) DHHS should serve as a catalyst bringing together, on a continuing basis, concerned groups focused on specific issues, such as cholesterol, cigarette smoking, and worksite health with specific attention given to minority issues. The National High Blood Pressure Education Program could serve as a model for this activity.
2) The formation of subcommittees to major task forces or standing committees within DHHS should be considered and given the charge of maintaining awareness of minority health issues and of facilitating the implementation and monitoring of initiatives resulting from this Task Force report. In addition, DHHS should take advantage of minority health professionals, administrators, and other staff who might aid in carrying out and monitoring initiatives emanating from the Task Force report.
3) The Federal government should serve as a model of minority workplace health policies, for example, by offering preventive health services and by networking these policies with the health care provider industry.

\section*{H. Data Issues}
1) Substantial oversampling in the major national NCHS surveys of minorities, Blacks and Hispanics in particular, is recommended. Because such an approach for Asian subgroups and Native Americans is not feasible, other directed approaches, such as cohort studies should be considered.
2) Methods of tracking major health problems, particularly nonfatal as well as fatal ones, in minority groups are needed. The primary goal of such studies would be to obtain feedback as rapidly as possible.
This would enable early recognition and treatment of adverse trends in health status.
3) The timely completion of the Hispanic HANES and the dissemination of the results is recommended to provide more reliable and valid estimates of cardiovascular disease morbidity in a large and representative sample of Mexican Americans, Puerto Ricans, and

Cubans. Information on the other Hispanic Americans would also be useful even though their numbers are comparatively smaller and they are widely dispersed throughout the United States.
4) Longitudinal follow-up of NHANES is recommended as well as a reduction of the examination interval from 4 to 2 years is recommended.
5) Increased analytical activities for data already collected by DHHS is recommended. These analyses should not be done to the detriment of continued collection of data.
6) Existing NCHS data bases should be examined to separate grouped data into specific minorities where feasible, namely Blacks (not Hispanics), Whites (not Hispanics), Hispanics, Puerto Rican Americans, Cuban Americans, Mexican Americans, Asian/Pacific Islander Americans, Japanese Americans, Chinese Americans, Korean Americans, Filipino Americans and Native Americans. In addition, where feasible, a breakdown of these data by urban vs. rural residency, geographic location, socioeconomic status, age, and gender is recommended.
7) Health statistics need to be developed on growing subgroups of other Asians such as Polynesians, Cambodians, Vietnamese, and East Indians. These groups constitute distinct cultural entities, and many of these populations are likely to be at disproportionate risk for a variety of illnesses due to their refugee status, endemic poverty, and/or other sociocultural factors.
8) Consideration of economic and cultural factors such as nativity, geographic area of residency, level of acculturation, and socio-economic status and mobility should be considered in analyses of data from minorities.
9) Techniques to improve collection of census data for minorities need to be developed.
10) Approaches for improving death certificate diagnosis and reporting procedures for Blacks and, presumably, other minorities need to be developed.
11) The geographic locations in which Black cohort studies of CHD incidence are performed need to be expanded.
12) Success in reducing the prevalence of substance abuse, violence, and vehicular accidents in Native American populations, which will increase life expectancy, can be expected to increase the prevalence and mortality risk from cardiovascular diseases and cancer in these groups. Therefore, it is important to monitor CHD mortality trends paying close attention to changes across tribes, regions, age and gender groups, and in Indians
age and gender groups, and in Indians living in rural
living in rural locations vs. those who have migrated to urban centers.
13) Small group-specific cohort-controlled studies to determine more accurately the actual CHD mortality, morbidity, and risk factor status of Native Americans are recommended. Specific emphasis should be placed on monitoring these disease trends over time in the more high-risk subgroups in the major tribes.

\section*{REFERENCES}

\section*{Black Americans}
1. Rice DP, Feldman JJ, White KL. The Current Burden of Illness in the United States. Institute of Medicine Occasional Paper, Number 3, October 27, 1976, Washington, DC.
2. Gillum RF. Coronary Heart Disease in Black Populations: I. Mortality and Morbidity. American Heart Journal 104(4):839-851, 1982.
3. Data provided to Task Force by National Center for Health Statistics.
4. Vital Statistics of the United States, National Center for Health Statistics.
5. National Heart, Lung, and Blood Institute: Conference on the Decline in Coronary Heart Disease Mortality, Report of Proceedings. DHEW Pub. No. (NIH) 79-1610. National Institutes of Health. Washington. US Government Printing Office, 1979.
6. Gillum RF, Folsom AR, Blackburn H. Decline in Coronary Heart Disease Mortality: Old Questions and New Facts. American Journal of Medicine 76(6):1055-1065, 1984.
7. Gillum RF, Liu KC. Coronary Heart Disease Mortality in United States Blacks, 1940-1978: Trends and Unanswered Questions. American Heart Journal 108(3;2): 728-732, 1984.
8. Watkins LO. Epidemiology of Coronary Heart Disease in Black Populations: Methodologic Proposals. American Heart Journal 108(3;2):635-640, 1984.
9. National Center for Health Statistics: Coronary Heart Disease in Adults. US NCHS Pub. No. 1000, Series 11, No. 10. Washington, DC, 1965, US Government Printing Office.
10. Henderson \(M\), Savage DD. Prevalence and Incidence of Ischemic Heart Disease in US Black and White Populations. Commissioned Paper for the DHHS Task Force on Black and Minority Health, 1984-85.
11. Report of the Working Group on Arteriosclerosis of the National Heart, Lung, \& Blood Institute: Arteriosclerosis, 1981, Volume 2, NIH Publication No. 82-2035, Washington, DC, 1982, US Department of Health and Human Services, pages 178-180.
12. Langford HG, Oberman A, Borhani NO, Entwisle G, Tung B. Black-White Comparison of Indices of Coronary Heart Disease and Myocardial Infarction in the stepped care Cohort of the Hypertension Detection and Follow-up Program. American Heart Journal 108(3;2): 797-801, 1984.
13. Cassel J, Heyden S, Bartel AG, Kaplan BH, Tyroler HA, Cornoni JC, Hames CG. Incidence of Coronary Heart Disease by Ethnic Group, Social Class, and Sex. Archives of Internal Medicine 128(6): 901-906, 1971.
14. Tyroler HA, Knowles MG, Wing SB, Logue EE, Davis CE, Heiss G, Heyden S, Hames CG. Ischemic Heart Disease Risk Factors and 20 -year Mortality in Middle Age Evans County Black Males. American Heart Journal 108(3;2): 738-746, 1984.
15. Keil JE, Loadholt CD, Weinrich MC, Sandifer SH, Boyle E, Jr. Incidence of Coronary Heart Disease in Blacks in Charleston, SC American Heart Journal 108(3;2): 779-786, 1984.
16. Oalmann MC, McGill HL, Strong JP. Cardiovascular Mortality in a Community: Results of a Survey in New Orleans, American Journal of Epidemiology 94(6):546-555, 1971.
17. Oalmann MC, McGill HC, Deupree RH. Cardiovascular Mortality in a Community: Methodology and Objective Diagnostic Criteria. American Journal of Epidemiology 94(6):531-545, 1971.
18. Kuller L. Sudden Death in Atherosclerotic Heart Disease: The Case for Preventive Medicine. American Journal of Cardiology 24(5):617-628, 1969.
19. Hagstrom RM, Federsteil CF, Ho WY. Incidence of Myocardial Infarction and Sudden Death from Coronary Heart Disease in Nashville, TN, Circulation 44(5):884-890, 1971.
20. Kuller L, Perper J, Cooper M. Demographic Characteristics and Trends in Atherosclerotic Heart Disease Mortality: Sudden Death and Myocardial Infarction. Circulation 52(6):Supplement III, III-1-15, 1975.
21. Ahmed SS, Rozefort R, Brancato R. Incidence of Acute Myocardial Infarction Among Blacks in an Urban Community. Journal of the Medical Society of New Jersey 74:1058-1060, 1977.
22. Weisse \(A B\), Abiuso PD, Thind IS. Acute Myocardial Infarction in Newark, New Jersey: A Study of Racial Incidence. Archives of Internal Medicine 137:1402, 1977.
23. Kuller L, Cooper M, Perper J, Fisher R. Myocardial Infarction and Sudden Death in an Urban Community. Bulletin of the New York Academy of Medicine 49(6):532-543, 1973.
24. Zmyslinski RW, Lackland DT, Keil JE, Higgins JE. Increased Fatality and Difficult Diagnosis of In-Hospital Acute Myocardial Infarction: Comparison to Lower Mortality and More Easily Recognized Pre-Hospital Infarction. American Heart Journal 108(5):586-592, 1981.
25. National Center for Health Statistics, data from the Hospital Discharge Survey, 1981.
26. National Center for Health Statistics, Michael Rowland and Gene Roberts: Blood Pressure Levels and Hypertension in Persons Ages 6-74 Years: United States, 1976-80. Advance Data from Vital and Health Statistics, No. 84. DHHS Publication No. (PHS) 82-1250. Public Health Service. Hyattsville, Maryland, 1982.
27. Comstock GW. An Epidemiologic Study of Blood Pressure Levels in a Biracial Community in the Southern United States. American Journal of Hygiene 64:271-315, 1957.
28. National Center for Health Statistics: Blood Pressure of Adults By Age and Sex, United States, 1960-62. Vital and Health Statistics. PHS Publication No. 1000-series 11-No. 4. Public Health Service. Washington DC. US Government Printing Office, June, 1964.
29. National Center for Health Statistics: Hypertension in Adults 25-74 Years of Age. United States, 1971-1975. Vital and Health Statistics. Series 11, No. 221. DHHS Pub. No(PHS). 81-1671. Public Health Service. Washington, DC. U.S. Government Printing Office, April, 1981.
30. National Center for Health Statistics: Blood Pressure Levels of Persons 6-74 Years, United States, 1971-74. Vital and Health Statistics. PHS Publication No. 1000-Series 11 - No. 203. Public Health Service. Washington DC. US Government Printing Office, September 1977.
31. National Center for Health Statistics: Blood Pressure Levels in Persons 18-74 Years of Age in 1976-80, and Trends in Blood Pressure from 1960-1980 in the United States. Data from the National Health Survey, Series 11, forthcoming. DHHS Publication Number (PHS) 85- , Hyattsville, Maryland, 1985.
32. Freeman JL, Freeman DH Jr, Ostfeld AM. Analysis of Data from Successive Complex Sample Surveys, with an Example of Hypertension Prevalence from the United States Health Examination Survey. International Journal of Epidemiology 12(2):230-237, 1983.
33. Kuller LH. Stroke Report. Commissioned Paper prepared for the Task Force on Black and Minority Health, 1984-85.
34. Neaton JD, Kuller LH, Wentworth D, Borhani NO. Total and Cardiovascular Mortality in Relation to Cigarette Smoking, Serum Cholesterol Concentration, and Diastolic Blood Pressure among Black and White Males Followed up for Five Years. American Heart Journal 108(3;2): 759-770, 1984.
35. Stolley PD, Kuller LH, Nefzger MD, Tonascia S, Lilienfeld AM, Miller GD, Diamond EL. Three-area Epidemiological Study of Geographic Differences in Stroke Mortality. II. Results. Stroke 8(5):551-557, 1977.
36. Wylie CM. Recent Trends in Mortality From Cerebrovascular Accidents in the United States. Journal of Chronic Diseases 14:213-220, 1961.
36a. Kuller L, Seltser R, Paffenbarger RS, Kreuger DE. Trends in Cerebrovascular Disease Mortality Based On Multiple Cause Tabulation of Death Certificates, 1930-1960. American Journal of Epidemiology 88(3):307-317, 1968.
37. National Health Interview Survey 1972. As cited in Kuller LH. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85.
38. Hypertension Detection and Follow-up Program Cooperative Group. Five Year Findings of the Hypertension Detection and Follow-up Program. III. Reduction in Stroke Incidence Among Persons with High Blood Pressure. Journal of the American Medical Association 247(5):633-638, 1982.
39. Heyman A, Karp HR, Heyden S, Bartel A, Cassel JC, Tyroler HA, Hames CG. Cerebrovascular Disease in the Biracial Population of Evans County, Georgia. Archives of Internal Medicine 128(6):949-955, 1971.
40. Gross CR, Kase CS, Mohr JP, Cunningham SC, Baker WE. Stroke in South Alabama: Incidence and Diagnostic Features - a Population-Based Study. Stroke 15(2):249-255, 1984.
41. Burton BT, Hirschman GH. Demographic Analysis: End-Stage Renal Disease and its Treatment in the United States. Clinical Nephrology 11(2):47-51, 1979.
42. Health Care Financing Administration, End-Stage Renal Disease Patient Profile Tables, 1981 and 1982. Department of Health and Human Services.
43. Easterling RE. Racial Factors in the Incidence and Causation of End-Stage Renal Disease. Transactions of the American Society for Artificial Internal Organs 23:28-33, 1977.
44. Rostand SG, Kirk KA, Rutsky EA, Pate BA. Racial Differences in the Incidence of Treatment for End-Stage Renal Disease. New England Journal of Medicine 306(May 27):1276-1279, 1982.
45. Sugimoto T, Rosansky SJ. The Incidence of Treated End-Stage Renal Disease in the Eastern United States: 1973-1979. American Journal of Public Health 74(1):14-17, 1984.
46. Krakauer H, Grauman JS, McMullan MR, and Creede CC. The Recent U.S. Experience in the Treatment of End-Stage Renal Disease By Dialysis and Transplantation. New England Journal of Medicine 308(June 30):1558-1563, 1983.
47. Cooper RS, Steinhauer MJ, Miller WJ et al. Racism, Society and Disease: An Exploration of the Social and Biological Mechanisms of Differential Mortality. International Journal of Health Services 11:389-414, 1981.
48. Gillum RF, Grant CT. Coronary Heart Disease in Black Populations II. Risk Factors. American Heart Journal 104(4;1): 852-864, 1982.
49. Durant RH, Linder CW, Jay S, Harkness JW, Gray RS. The Influence of a Family History of CHD Risk Factors on Serum Lipoprotein Levels in Black Children and Adolescents. Journal of Adolescent Health Care 3(2):75-81, 1982.
50. Haywood LJ. Coronary Heart Disease Mortality/Morbidity and Risk in Blacks. I. Clinical Manifestations and Diagnostic Criteria: the Experience with the Beta Blocker Heart Attack Trial. American Heart Journal 108(3;2): 787-793, 1984.
51. Hypertension Detection and Follow-up Program Cooperative Group. Effect of stepped care Treatment on the Incidence of Myocardial Infarction and Angina Pectoris: Five Year Findings of the Hypertension Detection and Follow-up Program. Hypertension 6(Supplement I):I-198-I-206, 1984.
52. National Center for Health Statistics: Serum Cholesterol Levels of Adults: United States, 1960-62. US Vital and Health Statistics, Series 11, No. 22, USDHEW Publication No. (HRA) 77-1283. Washington, DC, 1967, US Government Printing Office.
53. National Center for Health Statistics: Total Serum Cholesterol Levels of Adults 18-74 Years: United States, 1971-74. US Vital and Health Statistics, Series 11, Number 205, USDHEW No. (PHS) 78-1652. Washington, DC, 1978, US Government Printing Office.
54. National Center for Health Statistics: Serum Cholesterol Levels of Persons 4-74 Years of Age by Socioeconomic Characteristics, United States, 1971-74. US Vital and Health Statistics, Series 11, Number 217, USDHEW Publication Number (PHS) 80-1667, Washington, DC, 1980, US Government Printing Office.
55. Rowland ML, Fulwood R. Coronary Heart Disease Risk Factor Trends in Blacks Between the First and Second National Health and Nutrition Examination Surveys, United States, 1971-1980. American Heart Journal 108(3;2): 771-779, 1984.
56. Tyroler HA, Glueck CJ, Christensen B, Kwiterovich PO. Plasma High Density Lipoprotein Cholesterol; Comparisons in Black and White Populations. Circulation 62(Supplement 4):99-107, 1980.
57. Glueck CJ, Gartside P, Laskarzewski PM, Khoury P, Tyroler HA. A High Density Lipoprotein Cholesterol in Blacks and Whites: Potential Ramifications for Coronary Heart Disease. American Heart Journal 108(3;2): 815-826, 1984.
58. Gartside PS, Khoury P, Glueck CJ. Determinants of High Density Lipoprotein Cholesterol in Blacks and Whites: The Second National Health and Nutrition Examination Survey. American Heart Journal 108(3;2): 641-653, 1984.
59. Tyroler HA, Hames CG, Krishan I, Heyden S, Cooper G, Cassel JC. Black-White Differences in Serum Lipids and Lipoproteins in Evans County. Preventive Medicine 4:541-549, 1975.
60. Kleinbaum DG, Kupper LL, Cassel JC, Tyroler HA. Multivariate Analysis of Risk of Coronary Heart Disease in Evans County, Georgia. Archives of Internal Medicine 128(6):943-948, 1971.
61. Kleinman JC, Feldman JJ, Monk MA. The Effects of Changes in Smoking Habits on Coronary Heart Disease Mortality. American Journal of Public Health 69(8):795-802, 1979.
62. Schoenborn CA, Danchik KM. Health Practices Among Adults, United States, 1977. National Center for Health Statistics. Advance Data No. 64. November 4, 1980.
63. Moss AJ. Changes in Cigarette Smoking and Current Smoking Practices Among Adults. United States, 1978. National Center for Health Statistics. Advance Data No. 52. September 19, 1979.
64. Garfinkel L. Cigarette Smoking and Coronary Heart Disease in Blacks: Comparison to Whites in a Prospective Study. American Heart Journal, 108(3):Part II, 802-807, 1984.
65. National Center for Health Statistics: Characteristics of Persons with Diabetes. United States: July 1964-June 1965. US Vital and Health Statistics, Series 10, No. 40. Washington, DC, 1967, United States Printing Office.
66. National Center for Health Statistics: Prevalence of Chronic Conditions of the Genitourinary, Nervous, Endocrine, Metabolic and Blood and Blood-Forming Systems and of Other Selected Chronic Conditions. US Vital and Health Statistics, Series 10, No. 109, Washington, DC, 1973, US Government Printing Office.
67. Cooper R, Liu K, Stamler J, Schoenberger JA, Shekelle RB, Collette P, Shekelle S. Prevalence of Diabetes/Hyperglycemia and Associated Cardiovascular Risk Factors in Blacks and Whites: Chicago Heart Association Detection Project in Industry. American Heart Journal 108(3;2): 827-833, 1984.
68. Dales LG, Siegelaub AB, Feldman R, Friedman GD, Seltzer CC, Collen MF. Racial Differences in Serum and Urine Glucose After Challenge. Diabetes 23:327-332, 1974.
69. Stamler J, Wentworth D, Neaton J, Schoenberger JA, Feigal D. for the MRFIT Reseach Group, Diabetes and Risk of Coronary, Cardiovascular, and All Causes Mortality: Findings for 356,000 Men Screened by the Multiple Risk Factor Intervention Trial. (MRFIT). Circulation 70(Supplement 2):II-161, 1984.
70. Cedres BL, Liu K, Stamler J, Dyer AR, Stamler R, Berkson DM, Paul O, Lepper M, Linberg HA, Marquardt J, Stevens E, Schoenberger JA, Shekelle RB, Collette P, Garside D. Independent Contribution of Electrocardiographic Abnormalities to Risk of Death From Coronary Heart Disease, Cardiovascular Disease and All Causes: Findings of Three Chicago Epidemiologic Studies. Circulation 65(1):146-153, 1982.
71. Kannel WB, Gordon T, Castelli WP, Margolis JR. Electrocardiographic Left Ventricular Hypertrophy and Risk of Coronary Heart Disease: The Framingham Study. Annals of Internal Medicine 72(6):813-822, 1970.
72. Riley CP, Oberman A, Hurst DC, Peacock PB. Electrocardiographic Findings in an Biracial, Urban Population. The Birmingham Stroke Study. Alabama Journal of Medical Science 10:160-170, 1973.
73. Beaglehole R, Tyroler HA, Cassel JC, Deubner DC, Bartel A, Hames CG. An Epidemiological Study of Left Ventricular Hypertrophy in the Biracial Population of Evans County, Georgia, Journal of Chronic Diseases 28:554-559, 1975.
74. Bartel A, Heyden S, Tyroler HA, Tabesh E, Cassel JC, Hames CG. Electrocardiographic Predictors of Coronary Heart Disease. Archives of Internal Medicine 128(6):929-937, 1971.
75. Tyroler HA. Race, Education, and Five Year Mortality in HDFP Stratum 1 Referred Care Males. In Gross F, and Strasser T (Editors), Mild Hypertension: Recent Advances, Raven, New York, 1983, pp 163-176.
76. National Center for Health Statistics: Weight By Height and Age Of Adults, United States 1960-62. J Roberts. Vital and Health Statistics PHS Publication No. 1000; Series 11; No. 14. Public Health Service, Washington, DC. U.S. Government Printing Office. May 1966.
77. Weight and Height of Adults 18-74 Years of Age: United States, 1971-74. US Vital and Health Statistics, Series 11, No. 211. DHEW Publication No. (PHS) 79-1659, Washington, DC 1979, US Government Printing Office, Page 3.
78. Tyroler HA, Heyden S, Bartel A, Kassel J, Cornoni JC, Hames CG, Kleinbaum D. Blood Pressure and Cholesterol as Coronary Heart Disease Risk Factors. Archives of Internal Medicine 128(6): 907-914, 1971.
79. Gillum RF. Pathophysiology of Hypertension in Blacks and Whites: A Review of the Basis of Racial Blood Pressure Differences. Hypertension 1:468-475, 1979.
80. van Den Berghe P. Racialism and Assimilation in Africa and the Americas.Southwestern Journal of Anthropology 19:424-432, 1963.
81. Lewontin RC. The Apportionment of Human Diversity. Evolutionary Biology 6:381-398, 1973.
82. Cooper R. A Note on the Biologic Concept of Race and its Application in Epidemiologic Research. American Heart Journal 108(3;2): 715-723, 1984.
83. Reed TE. Caucasian Genes in American Negroes. Science 165: 762-768, 1969.
84. Williams AW. Blood Pressure of Africans. East African Medical Journal 18:109-117, 1941.
85. Shaper AG, Saxton GA. Blood Pressure and Body Build in a Rural Community in Uganda. East African Medical Journal 46:228-245, 1969.
86. Vaughan GP. Blood Pressure and Heart Murmurs in a Rural Population in the United Republic of Tanzania. Bulletin of the World Health Organization 57:89-97, 1979.
87. Ree GH. Arterial Pressures in a West African (Gambian) Rural Population. Journal of Tropical Medicine and Hygiene 76:65-70, 1973.
88. Pobee JOM, Larbi EB, Belcher DW, Wurapa FK, Dodu SRA. Blood Pressure Distribution in a Rural Ghanaian Population. Transactions of the Royal Society of Tropical Medicine and Hygiene 71:66-72, 1977.
89. Seedat YK, Seedat MA. An Interracial Study of the Prevalence of Hypertension in an Urban South African Population. Transactions of the Royal Society of Tropical Medicine and Hygiene 76:62-71, 1982.
90. Johnson BC, Remington RD. A Sampling Study of Blood Pressure Levels in White and Negro Residence of Nassau, Bahamas. Journal of Chronic Diseases 13:39-51, 1961.
91. Schneckloth RE, Corcoran AC, Stuart KL, Moore FE. Arterial Pressure and Hypertensive Disease in a West Indian Negro Population. American Heart Journal 63:607-628, 1962.
92. Miall WE, Kass EH, Ling J, Stuart KL. Factors Influencing Arterial Pressures in the General Population in Jamaica. British Medical Journal 2:497-508, 1962.
93. Florey C DuV, Ashcroft MT, Miller GJ. Blood Pressure Levels in Guyanese Adults of African and Indian Origin. American Journal of Epidemiology 94:419-424, 1971.
94. Khaw KT, Rose G. Population Study of Blood Pressure and Associated Factors in St. Lucia, West Indies. International Journal of Epidemiology 11:372-377, 1982.
95. Luft FC, Grim CE, Higgins JT Jr, Weinberger MH. Differences in Response to Sodium Administration in Normotensive White and Black Subjects. Journal of Laboratory and Clinical Medicine 90:555-562, 1977.
96. Grim CE, Luft FC, Miller JZ, Meneely Jr, Battarbee HD, Hames CG, Dah1 LK. Racial Differences in Blood Pressure in Evans County, Georgia: Relationship to Sodium and Potassium Intake and Plasma Renin Activity. Journal of Chronic Disease 33:87-94, 1980.
97. Watson RL, Langford HG, Abernethy J, Barnes TY, Watson MJ. Urinary Electrolytes, Body Weight and Blood Pressure. Pooled Cross Sectional Results among Four Groups of Adolescent Females. Hypertension 2(Supplement 1):93, 1980.
98. Frisancho AR, Leonard WR, Bollettino LA. Blood Pressure in Blacks and Whites and its Relationship to Dietary Sodium and Potassium Intake. Journal of Chronic Diseases 37:515-519, 1984.
99. Langford HG. Dietary Potassium and Hypertension. Annals of Internal Medicine 98:770-772, 1983.
100. Langford HG. Is Blood Pressure Different in Black People? Postgraduate Medical Journal 57:749-754, 1981.
101. Grell GAC, Forrester T, Robinson HM, Renin Levels in Hypertensive Patients in Jamaica. West Indian Medical Journal 30:30-33, 1981.
102. Blaustein MP. Sodium Transport and Hypertension: Where Are We Going? Hypertension 6:445-453, 1984.
103. Trevisan M, Ostrow D, Cooper RS, Sempos C, Stamler J. Sex and Race Differences in Sodium-Lithium Countertransport and Red Cell Sodium Concentration. American Journal of Epidemiology 120(4):537-541, 1984.
104. Ringell RE, Hamlyn JM, Schaeffer J, Hamilton BP, Kowarski AA, Blaustein MP, Berman MA. Red Cell Cotransport Activity and Sodium Content in Black Men. Hypertension 6(5):724-730, 1984.
105. Tuck ML, Gross C, Maxwell MH, Brickman AS, Krasnoshtein G, Mayes D. Erythrocyte Sodium Potassium Cotransport and Sodium Potassium Pump in Black and Caucasian Hypertensive Patients. Hypertension 6(4):536-544, 1984.
106. Johnson AL, Cornoni J, Cassel JC, Tyroler HA, Heyden S, Hames CG.Influence of Race, Sex and Weight on Blood Pressure Behavior In Young Adults. American Journal of Cardiology 35(4):523-530, 1975.
107. Dischinger PC, Apostolides AY, Entwisle G, Hebel JR. Hypertension Incidence in an Inner City Black Population. Journal of Chronic Diseases 34:405-413, 1981.
108. Voors AW, Dalferes ER Jr, Frank GC, Aristimuno GG, Berenson GS. Relation Between Ingested Potassium and Sodium Balance in Young Blacks and Whites. American Journal of Clinical Nutrition 37:583, 1983.
109. Kannel WB, Wolfe PA, Verter J. Manifestations of Coronary Disease Predisposing to Stroke: The Framingham Study. Journal of the American Medical Association 250(21):2942-2946, 1983.
110. Heyman A, Karp HR, Heyden S, Bartel A, Cassel JC, Tyroler HA, Hames CG. Cerebrovascular Disease in the Biracial Population of Evans County, Georgia. Archives of Internal Medicine 128:949-955, 1971.
111. Cooper R, Stamler J, Dyer A, Garside D. The Decline in Mortality from Coronary Heart Disease, USA, 1968-1975. Journal of Chronic Diseases 31:709-720, 1978.
112. Lindner A, Charra B, Sherrard DJ, Scribner BH. Accelerated Atherosclerosis in Prolonged Maintenance Hemodialysis. New England Journal of Medicine 290:697-701, 1974.
113. Goldberg AP, Harter HR, Patsch W, Schechtman KB, Province M, Weerts C, Kuisk I, McCrate MM, Schonfeld G. Racial Differences in Plamsa High Density Lipoproteins in Patients Receiving Hemodialysis: A Possible Mechanism for Accelerated Atherosclerosis in White Men. New England Journal of Medicine 308:1245-1252, 1983.
114. Hardy RJ, Hawkins CM. The Impact of Selected Indices of Antihypertensive Treatment on All-Cause Mortality. American Journal of Epidemiology 117(5):566-574, 1983.
115. Hypertension Detection and Follow-up Program Cooperative Group. Five Year Findings of the Hypertension Detection and Follow-up Program: Mortality By Race, Sex and Blood Pressure Level. A Further Analysis. Journal of Community Health 9(4):314-327, 1984.
116. Langford HG. Reduced Stroke Incidence in HDFP Enhanced By Strong Support System, Letter to the Editor. American Heart Journal 103(6):1087-1088, 1982.
117. Hypertension Detection and Follow-up Program Cooperative Group Five Year Findings of the Hypertension Detection and Follow-up Program: I. Reduction in Mortality of Persons With High Blood Pressure, Including Mild Hypertension. Journal of the American Medical Association 242(23):2562-2571, 1979.
118. Wing S. The Role of Medicine in the Decline of HypertensionRelated Mortality. International Journal of Health Services 14(4):649-666, 1984.
119. Wing S, Manton KG, The Contribution of Hypertension to Mortality in the US: 1968, 1977. American Journal of Public Health 73(2):140-144, 1983.
120. The Socioeconomic Status of the Black Population in the United States, 1973. Current Population Reports, Special Studies, Series p-23, No. 48, Washington, DC, July, 1974, US Bureau of the Census.
121. A Dream Deferred: The Economic Status of Black Americans, a Working Paper by the Center for the Study of Social Policy, Washington, DC, July 1983.
122. The Social and Economic Status of the Black Population in the United States: An Historical View, 1790-1978, Current Population Reports, Special Studies, Series p-23, No. 30, United States Department of Commerce, Bureau of the Census.
123. America's Black Population: 1970-1982, a Statistical View, Special Publication PIO/POP-83:1, US Department of Commerce, Bureau of the Census, July, 1983.
124. The State of Black America, 1985. A Report of the National Urban League, Washington, DC, January 1985.
125. Reimers CW, Sources of the Family Income Differentials Among Hispanics, Blacks and White Non-Hispanics. American Journal of Sociology 89(4):889-903, 1984.
126. Jencks C, Structural Versus Individual Explanations of Inequality: Where Do We Go From Here? Contemporary Sociology 9(6):762-767, 1980.
127. Antonovsky A, Social Class and the Major Cardiovascular Diseases. Journal of Chronic Diseases 21:65-106, 1968.
128. Jenkins CD, Psychosocial Risk Factors for Coronary Heart Disease. Acta Medica Scandinavica, Supplement 660:123-136, 1982.
129. Kasl SV. Social and Psychologic Factors in the Etiology of Coronary Heart Disease in Black Populations: An Exploration of Research Needs. American Heart Journal 108(3;2): 660669, 1984.
130. James SA. Socioeconomic Influences on Coronary Heart Disease in Black Populations. American Heart Journal 108(3;2): 669672, 1984.
131. James SA, Coronary Heart Disease in Black Americans: Suggestions For Research on Psychosocial Factors. American Heart Journal 108(3;2): 833-838, 1984.
132. Cassel JC, Heyden S, Bartel AG, Kaplan BH, Tyroler HA, Cornoni JC, Hames CG. Incidence of Coronary Heart Disease by Ethnic Group, Socicl Class and Sex. Archives of Internal Medicine 128:901-906, 1971.
133. Ruberman W, Weinblatt E, Goldberg JD, Chaudhary BS. Psychosocial Influences on Mortality After Myocardial Infarction. New England Journal of Medicine 311:552-559, 1984.
134. Kraus JF, Borhani NO, Franti CE. Socioeconomic Status, Ethnicity and Risk of Coronary Heart Disease. American Journal of Epidemiology 111(4):407-414, 1980.
135. Wilson PWF, Savage DD, Castelli WP, Garrison RJ, Donohue RT, Feinleib F. HDL-Cholesterol in a Sample of Black Adults: The Framingham Minority Study. Metabolism 32(4):328-332, 1983.
136. Khoury PR, Morrison JA, Laskarzewski P, Kelly K, Mellies MJ, King P, Larsen R, Glueck CG. Relationships of Education and Occupation to Coronary Heart Disease Risk Factors in School Children and Adults. The Princeton School District Study. American Journal of Epidemiology 113(4):378-395, 1981.
137. Hypertension Detection and Follow-up Program Cooperative Group. Race, Education and Prevalence of Hypertension. American Journal of Epidemiology 106(5):351-361, 1977.
138. Shulman NB, Martinez B, Brogan DR, Carr AA, Miles CG. Financial Cost as an Obstacle to Hypertension Therapy. (Submitted for Publication).
139. Syme LS, Oakes TW, Friedman GD, Feldman R, Siegelaub AB, Collins M. Social Class and Racial Differences in Blood Pressure. American Journal of Public Health 64:619-620, 1974.
140. Keil JE, Tyroler HA, Sandifer SH, Boyle E Jr. Hypertension: Effects of Social Class and Racial Admixture: The Results of a Cohort Study in the Black Population of Charleston, South Carolina. American Journal of Public Health 67(7):634-639, 1977.
141. Harburg E, Erfurt JC, Chape C, Hauenstein LS, Schull WJ, Schork MA. Socioecological Stressor Areas and Black-White Blood Pressure: Detroit. Journal of Chronic Diseases 26:595-611, 1973.
142. Harburg E, Erfurt JC, Hauenstein LS, Chape C, Schull WJ, Schork MA. Socioecological Stress, Suppressed Hostility, Skin Color, and Black-White Male Blood Pressure: Detroit. Psychosomatic Medicine 35(4):276-296, 1973.
143. Harburg E, Gleibermann L, Roeper P, Schork MA, Schull WJ. Skin Color, Ethnicity, and Blood Pressure, I. Detroit Blacks. American Journal of Public Health 68(12):1177-1183, 1978.
144. Tyroler HA, James SA. Blood Pressure and Skin Color. American Journal of Public Health 68:1170-1172, 1978.
145. James SA, Hartnett SA, Kalsbeek WD. John Henryism and Blood Pressure Differences Among Black Men. Journal of Behavioral Medicine 6(3):259-278, 1983.
146. James SA, Lacroix AZ, Kleinbaum DG, Strogatz BS. John Henryism and Blood Pressure Differences Among Black Men.
II. The Role of Occupational Stressors. Journal of Behavioral Medicine 7(3):259-275, 1984.
147. Howard J, Holman BL. The Effects of Race and Occupation on Hypertension Mortality. Milbank Memorial Quarterly 48: 263-276, 1970.
148. Neser WB, Tyroler HA, Cassel JC. Social Organization and Stroke Mortality in the Black Population of North Carolina. American Journal of Epidemiology 93(3):166-175, 1971.
149. James SA, Kleinbaum DG. Socioecologic Stress and HypertensionRelated Mortality Rates in North Carolina. American Journal of Public Health 66(4):354-358, 1976.
150. Williams PB. Assessing Awareness of Coronary Disease Risk Factors in the Black Community. Urban Health 8(9):34-37, 1979.
151. The Public and High Blood Pressure: Six Year Follow-up Survey of Public Knowledge and Reported Behavior. US Department of Health and Human Services. NIH Publication No. 81-2118, September 1981.
152. Heimbach JT. Diet and Cardiovascular Disease: The Public View. Public Health Reports. January 1985.
153. Sunseri AJ, Alberti JM, Kent ND, Dolocek TA, Schoenberger JA. Ingredients in Nutrition Education: Family Involvement, Reading and Race. Journal of School Health 55(5):193-196, 1984.
154. Payne ZA. Diet and Cardiovascular Disease: The Morbid Connection. Journal of the Medical Association of Georgia, 74(1):49-52, 1985.
155. Wheeler M, Haider SQ. Buying and Food Preparation Patterns of Ghetto Blacks and Hispanics in Brooklyn. Journal of the American Dietetic Association 75:560-563, 1979.
156. Connett JE, Stamler J. Responses of Black and White Males to the Special Intervention Program of the Multiple Risk Factor Intervention Trial. American Heart Journal 108(3;2): 839-849, 1984.
157. Green LW. Diffusion and Adoption of Innovations Related to Cardiovascular Risk Behavior in the Public. Chapter in: Applying Behavioral Science to Cardiovascular Risk, Enelow AJ and Henderson JB (Editors), American Heart Association Inc. 1975, pp 84-108.
158. Covey LS, Mushinski MH, Wynder EL. Smoking Habits in a Hospitalized Population. American Journal of Public Health 73:1293-1297, 1983.
159. EVAXX, Inc. A Study of Smoking Behavior Among Black Americans, Presentation at Workshop on Smoking Prevention and Cessation for National Cancer Institute Division of Research Centers and Community Activities, March, 1983.
160. Baugh JG, Hunter SM, Webber LS, Berenson GS. Development Trends of First Cigarette Smoking Experiene of Children: The Bogalusa Heart Study. American Journal of Public Health 72(10): 1161-1164, 1982.
161. Ward WB, Levine DM, Morisky D, Bone LR, Ward E, Soff G, McKinney R. Controlling High Blood Pressure in Inner City Baltimore Through Community Health Education. Chapter in: Perspectives in Community Health Education: A Series of Case Studies, Carlaw RW (Editor), Third Party Publishing Co., Oakland, 1982, pp 73-97.
162. Wagner EH, Slone C, Carroll CL, Warner JT, Pittman AW, Pickard CG, Williams BO, Cornoni-Huntley JC. Hypertension Control in a Rural Biracial Community: Successes and Failures of Primary Care. American Journal of Public Health 70(1):48-55, 1980.
163. Kasl SV. Social-Psychological Characteristics Associated With Behaviors Which Reduce Cardiovascular Risk. Chapter in: Applying Behavioral Science to Cardiovascular Risk, Enelow AJ, Henderson JB, (Editors), American Heart Association, 1975, 173-190.
164. Working Group on Noncompliance in Black Male Hypertensives, Summary Report, Dynamic Programs, Washington, DC, April 1983.
165. Dutton DB. Explaining the Low Use of Health Services by the Poor: Costs, Attitudes or Delivery Systems? American Sociological Review 43:348-368, 1978.
166. Caldwell JR, Cobb S, Dowling M, Dejongh D. The Drop-Out Problem in Antihypertensive Treatment: A Pilot Study of Social and Emotional Factors Influencing a Patient's Ability To Follow Antihypertensive Treatment. Journal of Chronic Diseases 22:579-592, 1970.
167. Nelson EC, Stason WB, Neutra RR, Solomon HS. Identification of the Noncompliant Hypertensive Patient. Preventive Medicine 9:504-517, 1980.
168. Hypertension Detection and Follow-up Program Cooperative Group. Patient Participation in a Hypertension Control Program. Journal of the American Medical Association 239:1507-1514, 1978.
169. Smith EO, Curb JD, Hardy RJ, Hawkins CM, Tyroler HA. Clinic Attendance in the Hypertension Detection and Follow-up Program. Hypertension 4(5):710-715, 1982.
170. Hershey JC, Morton BG, Davis JB, Reichgott MJ. Patient Compliance With Antihypertensive Medication. American Journal of Public Health 70(19):1081-1089, 1980.
171. Cummings KM, Kirscht JP, Binder LR, Godley AJ. Determinants of Drug Treatment Maintenance Among Hypertensive Persons in Inner City Detroit. Public Health Reports 97(2):99-106, 1982.
172. Kasl SV. A Social-Psychological Perspective on Successful Community Control of High Blood Pressure: A Review. Journal of Behavioral Medicine 1(4):347-381, 1978.
173. Syme SL. Drug Treatment of Mild Hypertension: Social and Psychological Considerations. Annals of the New York Academy of Sciences 304:99-106, 1978.
174. Caldwell JR, Tiesen V, Kaunisto CA, Reddy PJ, Smythe PS, Smith BW. Psychosocial Factors Influence Control of Moderate and Severe Hypertension. Social Science and Medicine 17(12):773-782, 1983.
175. Morisky DE, Bowler MH, Finlay JS. An Educational and Behavioral Approach Toward Increasing Patient Activation in Hypertension Management. Journal of Community Health 7(3): 171-182, 1982.
176. Morisky DE, Levine DM, Greene LW, Shapiro S, Russel1 RP, Smith CR. Five Year Blood Pressure Control and Mortality Following Health Education for Hypertensive Patients. American Journal of Public Health 73(2):153-162, 1983.
177. Levine D. Health Education for Behavioral Change - Clinical Trial to Public Health Program. The Johns Hopkins Medical Journal 151:215-219, 1982.
178. Bowler MH, Morisky DE, Deeds SG. Needs Assessment Strategies in Working With Compliance Issues and Blood Pressure Control. Patient Counseling and Health Education 1:22-27, 1980.
179. Green LW, Levine DM, Deeds SG. Clinical Trials of Health Education for Hypertensive Patients: Design and Baseline Data. Preventive Medicine 4:417-425, 1975.
180. Whitehead TL, Frate LA, Johnson SA. Control of High Blood Pressure From Two Community-Based Perspectives. Human Organization 43(2):163-167, 1984.
181. Eckenfels EJ, Frate DA, Logan EW, et al. Endemic Hypertension In a Poor, Black Rural Community: Can It Be Controlled? Journal of Chronic Diseases 30:499-518, 1977.
182. Starfield B, Simborg D, Horne S, Yourtee S. Continuity and Coordination in Primary Care: Their Achievement and Utility. Medical Care 14:625-636, 1976.
183. Rogers DE, Blendon RJ, Maloney TW. Who Needs Medicaid? New England Journal of Medicine 307:13-18, 1982.
184. Davis K, Gold M, Makuc D. Access to Health Care for the Poor: Does the Gap Remain? Annual Review of Public Health 2:159-182, 1981.
185. Davis K, Rowland D. Uninsured and Underserved: Inequities in Health Care in the United States. Milbank Memorial Fund Quarterly 61(2):149-176, 1983.
186. Link CR, Long SH, Settle RF. Access to Medical Care Under Medicaid: Differentials By Race. Journal of Health Politics Policy and Law 7(2):345-365, 1982.
187. Rice MF, Joans W. Black Health Inequities and the American Health Care System. Health Policy and Education 3:195214, 1982.
188. Shorr GI, Nutting PA. A Population-Based Assessment of the Continuity of Ambulatory Care. Medical Care 15:455-464, 1977.
189. Nutting PA, Shorr GI, Burkhalter BR. Assessing the Performance of Medical Care Systems: A Method and Its Application. Medical Care 19:281-296, 1981.
190. Nutting PA, Burkhalter BR, Dietrich D, Helmick E. Relationship of Size and Payment Mechanism to System Performance in Eleven Medical Care Systems. Medical Care 20:676-690, 1982.
191. Nutting PA, Barrick JE, Logue SC. The Impact of a Maternal and Child Health Care Program on the Quality of Prenatal Care: An Analysis By Risk Group. Journal of Community Health 4:267-279, 1979.
192. Penchansky R, Thomas JW. The Concept of Access: Definition and Relationship to Consumer Satisfaction. Medical Care 19(2): 127-140, 1981.
193. National Center for Health Statistics, Office Visits for Diseases of the Circulatory System, the National Ambulatory Medical Care Survey, United States, 1975-1976, Series 13, No. 40, DHEW publication No. (PHS) 79-1791, January 1979.
194. Aday LA, Andersen RM. The National Profile of Access to Medical Care: Where Do We Stand? American Journal of Public Health 74(12):1331-1339, 1984.
194a. Himmelstein DU, Woolhandler S, Harnly M, Bader MB, Silber R, Backer HD, Jones AA. Patient Transfers: Medical Practice as Social Triage. American Journal of Public Health 74(5): 494-497, 1984.
194b. Thurow LC. Learning To Say "No". New England Journal of Medicine, 311:1569-1572, 1984.
194c. Schieff G. The "Dumping" Problem: No Insurance; No Admission". New England Journal of Medicine 312(23): 1522, 1985.
195. Goldberg RJ, Kennedy HL. The Influence of Race on Prognosis After Acute Myocardial Infarction: A Community-Wide Perspective. Journal of Cardiac Rehabilitation 3:195-201, 1983.
196. Shapiro S, Weinblatt E, Frank CW, Sager RV. Social Factors in the Prognosis of Man Following First Myocardial Infarction. Milbank Memorial Fund Quarterly 48:37-50, 1970.
197. Kottke TE, Young DT, McCall MM. Effect of Social Class on Recovery From Myocardial Infarction. Minnesota Medicine (August) 590-597, 1980.
198. Yelin EH, Kramer JS, Epstein WZ. Is Health Care Use Equivalent Across Social Groups? A Diagnosis-Based Study. American Journal of Public Health 73(5):563-571, 1983.
199. Detailed Diagnoses and Surgical Procedures For Patients Discharged From Short-Stay Hospitals, United States, 1979.
DHHS Publication Number (PHS) 83-1733. Washington, DC, 1983, National Center for Health Statistics, US Public Health Service.
200. NCHS Data on Surgical Procedures, 1982.
201. Oberman A, Cutter G. Issues in the Natural History and

Treatment of Coronary Heart Disease in Black Populations:
Surgical Treatment. American Heart Journal 108(3;2):
688-694, 1984.
202. Pearson TA. Risk Factors for Arteriographically Defined Coronary Artery Disease. Ph.D. Thesis Johns Hopkins University, Baltimore, Maryland, 1983.
203. Watkins L Jr., Gardner K, Gott V, Gardner TJ. Coronary Heart Disease and Bypass Surgery in Urban Blacks. Journal of the National Medical Association 75(4):381-383, 1983.
204. Office Visits For Hypertension, National Ambulatory Medical Care Survey, United States, January 1975-December 1976. Advance Data No. 28, April 28, 1978.
205. Office Visits by Black Patients, National Ambulatory Medical Care Survey: United States, 1975-1976. Advance Data No. 50, July 23, 1979.
206. National Center for Health Statistics. Medication Therapy in Office Visits for Hypertension: National Ambulatory Care Survey, 1980. Advance Data No.8. DHHS Publication No. (PHS) 82-1250, 1982.
207. Physician Visits: Volume and Interval Since Last Visit, United States, 1980. Data from the National Health Survey. Series 10, No.144. DHHS Publication No. (PHS) 83-1572, Table 16, p 50, 1983.
208. James SA, Wagner EH, Strogatz DS, Beresford SAA, Kleinbaum DG, Williams CA, Cutchin LM, Ibrahim MA. The Edgecombe County (NC) High Blood Pressure Control Program: II. Barriers to the Use of Medical Care Among Hypertensives. American Journal of Public Health 74(5):468-472, 1984.
209. Finnerty FA Jr, Shaw LW, Himmelsbach CK. Hypertension in the Inner City. II. Detection and Follow-up. Circulation 74: 76-78, 1973.
210. Wilber JA, Barrow JG. Reducing Elevated Blood Pressure. Minnesota Medicine 52:1303-1305, 1969.
211. Brook RH, Ware JE Jr, Rogers WH, Keiler EB, Davies AR, Donald CA, Goldberg CA, Lohr KN, Masthay PC, Newhouse JP. Does Free Care Improve Adults' Health? Results From a Randomized Controlled Trial. New England Journal of Medicine 309:1426-1434, 1983.
212. Lurie N, Ward NB, Shapiro MF, Brook RH. Termination From Medi-Cal - Does It Affect Health? New England Journal of Medicine 311:480-484, 1984.
213. Lurie N, Ward NB, Shapiro MF, Gallego C, Vaghaiwalla R, Brook RH. Termination From Medi-Cal: One Year Later. Presented at 1984 National Meeting of the Robert Wood Johnson Clinical Scholars Program, Howey-In-The-Hills, Florida. October, 1984.
214. Ware A, Henson MA, Wilber JA. National Conference on High Blood Pressure Control: Unresolved Issues; New Approaches. April 28-30, 1985. Chicago.
215. Rocheleau B. Black Physicians and Ambulatory Care. Public Health Reports 93(3):278-282, 1978.
300. Health and Prevention Profile: United States 1983, National Center for Health Statistics/PHS/DHHS. DHHS Publication No. (PHS) 84-1232, 1984.
301. Harwood A. Introduction. In: A. Harwood (Ed) Ethnicity and Medical Care. Harvard University Press, Cambridge, MA, 1981, pp 1-25.
302. Fisher DW. Socioeconomic Pathogenicity. Hospital Practice 15 (September):15-19, 1984.
303. Davis C, Haub C, Willette J. U.S. Hispanics: Changing the Faces of America. Population Bulletin 38:1-45, 1983.
304. Hazuda HP. Differences in Socioeconomic Status and Acculturation Among Mexican Americans and Risk for Cardiovascular Disease. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85.
305. Frerichs RR, Chapman JM, Nourjah P, Maes M. Cardiovascular Diseases in Los Angeles County, 1978-1981. American Heart Association, Greater Los Angeles Affiliate, Inc., 1984.
306. Schoen R, Nelson VF. Mortality By Cause Among Spanish-Surnamed Californians, 1969-1971. Social Science Quarterly 62:259-274, 1981.
307. Bradshaw BS, Fonner E. Jr. The Mortality of Spanish-Surnamed Persons in Texas, 1969-1971. In: Bean FD, Frisbie, WP (Eds), The Demography of Racial and Ethnic Groups. Academic Press, New York, 1978, pp 261-282.
308. Stern MP, Gaskill SP. Secular Trends in Ischemic Heart Disease and Stroke Mortality From 1970 to 1976 in Spanish-Surnamed and Other White Individuals in Bexar County, Texas. Circulation 58: 537-543, 1978.
309. Kautz JA, Bradshaw BS, Fonner E. Trends in Cardiovascular Mortality in Spanish-Surnamed, Other White, and Black Persons in Texas, 1970-1975. Circulation 64:730-735, 1981.
310. Kuller LH. Stroke Report. Paper commisssioned by the DHHS Task Force on Black and Minority Health, 1984-85.
311. Rosenthal M, Hazuda HP, Stern MP. Trends in Ischemic Heart Disease (IHD) Mortality Among Mexican Americans in Texas. Abstract submitted to the 25 th Conference on Cardiovascular Disease Epidemiology of the American Heart Association. March 7-9, 1985. Tucson, AZ
312. Castro FG, Baezconde-Garbanati L, Beltran H. Risk Factors For Coronary Heart Disease in Hispanic Populations: A Review. Hispanic Journal of Psychology, in press, 1985.
313. Kumanyika SK, Savage DD. Ischemic Heart Disease Risk Factors in Hispanic Americans. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85.
314. Stern MP, Gaskill SP, Allen CR, Garza V, Gonzalez JL, Waldrop RH. Cardiovascular Risk Factors in Mexican Americans in Laredo, Texas. I. Prevalence of Overweight and Diabetes on Distribution of Serum Lipids. American Journal of Epidemiology 113:546-555, 1981.
315. Stern MP, Haskell WL, Wood PD, Osann KE, King AB, Farquhar VW. Affluence and Cardiovascular Risk Factors in Mexican Americans and Other Whites in Three Northern California Communities. Journal of Chronic Diseases 28:623-636, 1975.
316. Friis R, Nanjundappa G, Pendergast TJ, Wesh M. Coronary Heart Disease Mortality and Risk Among Hispanics and Non-Hispanics in Orange County, California. Public Health Reports 96(5):418-422, 1981.
317. Kraus JF, Borhani NO, Franti CE. Socioeconomic Status, Ethnicity, and Risk of Coronary Heart Disease. American Journal of Epidemiology 111:407-414, 1980.
318. Stern MP, Rosenthal M, Haffner SM, Hazuda HP, Franco LJ. Sex Differences in the Effects of Sociocultural Status on Diabetes and Cardivvascular Risk Factors in Mexican Americans: The San Antonio Heart Study. American Journal of Epidemiology, in press, 1985.
319. Hazuda HP, Stern MP, Gaskill SP, Haffner SM, Gardner LI. Ethnic Differences in Health Knowledge and Behaviors Related to the Prevention and Treatment of Coronary Heart Disease. The San Antonio Heart Study. American Journal of Epidemiology 117:717-728, 1983.
320. Ailinger RL. Hypertension Knowledge in a Hispanic Community. Nursing Research 3:207-210, 1982.
321. Ramirez AG, Herrick KL, Weaver FJ. El Asesino Silencioso: A Methodology For Alerting the Spanish-Speaking Community. Urban Health, June 1981, pp 44-48.
322. James SA. Coronary Heart Disease in Black Americans: Suggestions for Research on Psychosocial Factors. American Heart Journal 108(3;2):833-838, 1984.
323. Kasl SV. Social and Psychologic Factors in the Etiology of Coronary Heart Disease in Black Populations: An Exploration of Research Needs. American Heart Journal 108(3;2):660-668, 1984.
324. Curry CL, Oliver J, Mumtaz FB. Coronary Artery Disease in Blacks: Risk Factors. American Heart Journal 108(3;2):653-657, 1984.
325. Garcia-Palmieri MR, Sorlie P, Tillotson J, Costas R, Cordero E, Rodriguez M. Relationship of Dietary Intake to Subsequent Coronary Heart Disease Incidence: The Puerto Rican Heart Health Program. American Journal of Clinical Nutrition 33:1818-1827, 1980.
326. Garcia-Palmieri MR, Costas R, Cruz-Vidal M, et al. Risk Factors and Prevalence of Coronary Heart Disease in Puerto Rico. Circulation 42:541-549, 1970.
327. Glueck CJ, Gartside P, Laskarzewski PM, Khoury P, Tyroler HA. High-Density Lipoprotein Cholesterol in Blacks and Whites: Potential Ramifications For Coronary Heart Disease. American Heart Journal 108(3;2):815-826, 1984.
328. Haffner SM, Gaskill SP, Hazuda HP, Gardner LI, Stern MP. Saturated Fat and Cholesterol Avoidance By Mexican Americans and Anglos: The San Antonio Heart Study. Clinical Research 30:237A, 1982 .
329. Hanis CJ, Ferrel RE, Barton SA, Aguilar L, Garza-Ibarra A, Tulloch BR, Garcia CA, Schull WJ. Diabetes Among Mexican Americans in Starr County, Texas. American Journal of Epidemiology 118:659-672, 1983.
330. Hazuda HP, Haffner S, Stern M, Rosenthal M, Franco L. Effects of Acculturation and Socioeconomic Status on Obesity and Glucose Intolerance in Mexican American Men and Women. American Journal of Epidemiology 120:494-502, 1984.
331. National Diabetes Data Group. Classification and Diagnosis of Diabetes Mellitus and Other Categories of Glucose Tolerance. Diabetes 28:1039-1057, 1979.
332. Stern MP, Gaskill SP, Hazuda HP, Gardner LI, Haffner SM. Does Obesity Explain Excess Prevalence of Diabetes Among Mexican Americans? Results of the San Antonio Heart Study. Diabetologia 24:272-277, 1983.
333. Gardner LI, Stern MP, Haffner SM, Gaskil1, SP, et al. Prevalence of Diabetes in Mexican Americans. Relationship to Percent of Gene Pool Derived From Native American Sources. Diabetes 33: 86-92, 1984.
334. Releford JH, Stern MP, Gaskil1 SP, Hazuda HP. Social Class, Admixture, and Skin Color Variation in Mexican Americans and Anglo Americans Living in San Antonio, Texas. American Journal of Physical Anthropology 61:97-102, 1983.
335. Roberts RE, Lee ES. Health Practices Among Mexican Americans: Further Evidence From the Human Population Laboratory Studies. Preventive Medicine 9:675-688, 1980.
336. Mueller WH, Joos SK, Hanis CL, Zavaleta AN, Eichner J, Schull WJ. The Diabetes Alert Study: Growth, Fatness, and Fat Patterning, Adolescence Through Adulthood in Mexican Americans. American Journal of Physical Anthropology 64:389-399, 1984.
337. Joos SK, Mueller WH, Hanis CL, Schull WJ. Diabetes Alert Study: Weight History and Upper Body Obesity in Diabetic and Nondiabetic Mexican American Adults. Annals of Human Biology 11:167-171, 1984.
338. Malina RM, Little BB, Stern MP, Gaskill SP, Hazuda HP. Ethnic and Social Class Differences in Selected Anthropometric Characteristics of Mexican American and Anglo American Adults: The San Antonio Heart Study. Human Biology 55:867-883, 1983.
339. National Center for Health Statistics. Overweight Adults in the United States. Advance Data. Vital and Health Statistics 51:1-9, 1979.
340. Garn SM. Ten State Nutrition Survey. Subscapular and Tricepts Fatfold Data. Total U.S.A. Center for Human Growth and Development, University of Michigan, Ann Arbor. Unpublished.
341. Holck SE, Warren CW, Rochat RW, Smith JC. Lung Cancer Mortality and Smoking Habits: Mexican American Women. American Journal of Public Health 72:38-42, 1982.
342. Marcus AC, Crane LA. Smoking Behavior Among Hispanics: A Preliminary Report. In: PF Engstrom, PN Anderson, LS Mortonson (Eds) Advances in Cancer Control: Epidemiology and Research. ErlanLiss, New York, 1984, pp 141-151.
343. Medina AS, Wallace HM, Ralph N, Goldstein H. Adolescent Health in Alameda County. Journal of Adolescent Health Care 2:175-182, 1982.
344. Sorlie PD, Garcia-Palmieri R, Costas R, Cruz-Vidal M, Havlik R. Cigarette Smoking and Coronary Heart Disease in Puerto Rico. Preventive Medicine 11:304-316, 1982.
345. Schreiber JM, Homiak JP. Mexican Americans. In: A. Harwood (Ed) Ethnicity and Medical Care. Harvard University Press, Cambridge, MA, 1981, pp 264-336.
346. Siegel JS, Passel JS. Coverage of the Hispanic Population of the United States in the 1970 Census: A Methodological Analysis. Current Population Reports, Special Reports Series P-23, No. 82, 1979.
347. Lurie N, Ward NB, Shapiro MF, Brook RH. Termination from MediCal: Does It Affect Health? Special Report. New England Journal of Medicine 311(7):480-484, 1984.
348. Yelin EH, Kramer JS, Epstein WV. Is Health Care Use Equivalent Across Social Groups? A Diagnosis-Based Study. American Journal of Public Health 73(5):563-571, 1983.
349. Brock RH, Ware JE, Rogers WH, et al. Does Free Care Improve Adults' Health? Results From a Randomized Controlled Trial. New England Journal of Medicine 309:1426-1434, 1983.
350. Davis K, Gold M, Makuc D. Access to Health Care for the Poor: Does the Gap Remain? Annual Review of Public Health 2:159-182, 1981.
400. Yu ESH, Chang CF, Liu WT, Kan SH. Asian-White Mortality Differentials: Is There Excess Death? Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85.
401. Kan SH, Liu WT. The Poverty Class of Asian Americans. Paper presented at the Eastern Sociological Society Annual Meetings, Boston, MA, March 8-10, 1984.
402. Gould-Martin K, Ngin C. Chinese Americans. In: A. Harwood (Ed.) Ethnicity and Medical Care. Harvard University Press, Cambridge, 1981.
403. Kagan A, Harris BR, Winkelstein W, Johnson H, Kato SL, et al. Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Demographic, Physical, Dietary, and Biochemical Characteristics. Journal of Chronic Diseases 27:345-364, 1974.
404. Winkelstein W, Kagan A, Kato H, et al. Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Blood Pressure Distributions. American Journal of Epidemiology 102:502-513, 1975.
405. Reed D, McGee D, Yano K. Trends of Coronary Heart Disease Among Men of Japanese Ancestry in Hawaii. Journal of Community Health 8:149-159, 1983.
406. Marmot MG, Syme SL. Acculturation and Coronary Heart Disease in Japanese Americans. American Journal of Epidemiology 104:225-247, 1976.
407. Chapman JM, Frerichs RR, Maes EF. Cardiovascular Diseases in Los Angeles, 1980. American Heart Association, Greater Los Angeles Affiliation, Inc., 1983.
408. Frerichs RR, Chapman JM, Nourjah P, Maes M. Cardiovascular Diseases in Los Angeles County, 1979-1981. American Heart Association, Greater Los Angeles Affiliate, Inc., 1984.
409. King H. Selected Epidemiologic Aspects of Major Disease and Causes of Death Among Chinese in the United States and Asia. In: A. Kleinman, P Kunstadted, ER Alexander, JL Gale (Eds) Medicine in Chinese Cultures: Comparative Studies of Health Care in Chinese and Other Societies. DHEW Publication No. (NIH) 75-653, pp 487-550, 1975.
410. Bennett CG, Tokuyama GH, McBride TC. Cardiovascular-Renal Mortality in Hawaii. American Journal of Public Health 52:1418-1431, 1976.
411. Gerber LM, Madhavan S. Epidemiology of Coronary Heart Disease in Migrant Chinese Populations. Medical Anthropology 4:307-320, 1980.
412. Marmot MG, Syme LS, Kagan A, Kato H, Cohen JB, Belsky J. Epidemiological Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Prevalence of Coronary and Hypertensive Heart Disease and Associated Risk Factors. American Journal of Epidemiology 102(6):514-525, 1975.
\begin{tabular}{|c|c|}
\hline & Wenkham NS, Wolff RJ. A Half-Century of Changing Food Habits Among Japanese in Hawaii. Journal of the American Dietetic Asociation 57:29-32, 1970. \\
\hline 4 & Nichaman MZ, Hamilton \(H B\), Kagan A, Grier R, Sacks ST, Syme SL. Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Distribution of Biochemical Risk Factors. American Journal of Epidemiology 102:491-501, 1975. \\
\hline 415. & Kagan A, Gordon T, Rhoads GG, Schiffman JC. Some Factors Related to Coronary Heart Disease Incidence in Honolulu Japanese Men: The Honolulu Heart Study. International Journal of Epidemiology 4:271-279, 1975. \\
\hline & Takeya Y, Popper JS, et al. Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Incidence of Stroke in Japan and Hawaii. Stroke 15:15-23, 1984. \\
\hline & Yano K, Reed DM, McGee DL. Ten-Year Incidence of Coronary Heart Disease in the Honolulu Heart Program. Relationship to Biological and Lifestyle Characteristics. American Journal of Epidemiology 119:653-666, 1984. \\
\hline & Gordon T, Garcia-Palmieri MR, Kagan A, Kanne1 WB, Schiffman J. Differences in Coronary Heart Disease in Framingham, Honolulu, and Puerto Rico. Journal of Chronic Diseases 17:328-344, 1974. \\
\hline & Stavig GR, Igra A, Leonard AR. Hypertension Among Asians and Pacific Islanders in California. American Journal of Epidemiology 119:677-691, 1984. \\
\hline 420. & Igra A, Stavig GR, Leonard AR, et al. Hypertension and Related Health Problems in California. Results From the 1979 California Hypertension Survey. Hypertension Clearinghouse. Department of Health Services. Hypertension Control Program. 714 P Street, Sacramento, CA 95814. \\
\hline 421. & Kumanyika SK, Savage DD. Ischemic Heart Disease Risk Factors in Asian Americans. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85. \\
\hline 422 & Robertson TL, Kaot H, Gordon T, Kagan A, Rhoads GC, Land CE, Worth RM, Belsky J, et al. Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California: Coronary Heart Disease Risk Factors in Japan and Hawaii. American Journal of Cardiology 39:244-249, 1977. \\
\hline 423. & Gordon T, Kagan A, Garcia-Palmieri M, Kannel WB, Zukel WJ, Tillotson J, Sorlie P, Hjortland M. Diet and Its Relation to Coronary Heart Disease and Death in Three Populations. Circulation 63:500-515, 1981. \\
\hline 424 & Basset DR, Schroffner WG. Coronary Heart Disease in Chinese Men in Hawaii. Achives of Internal Medicine 125:478-487, 1970. \\
\hline 425 & Kuller LH. Stroke Report. Paper commissioned by the DHHS Task Force on Black and Minority Health, 1984-85. \\
\hline 426. & Reed D, McGee D, Yano K, Feinleib M. Social Networks and Coronary Heart Disease Among Japanese Men in Hawaii. American Journal of Epidemiology 117:284-396, 1983. \\
\hline
\end{tabular}
500. Berry JW. Acculturative Stress in Northern Canada: Ecological Cultural, and Psychologic Factors. In: R. Sheppard (Ed) Proceedings of the Third International Symposium, pp 490-497, 1974.
501. Heidenreich CA. Alcohol and Drug Use and Abuse Among American Indians: A Review of Issues and Sources. Journal of Drug Issues 6(3):256-272, 1976.
502. Littman G. Alcoholism, Illness, and Social Pathology Among American Indians in Transition. American Journal of Public Health 60(9):1787-1796, 1978.
503. Wintrob R, Daimen S. The Impact of Culture Change on Misstassini Cree Youth. Canadian Psychology Association Journal 19: 331-341, 1974.
504. Health of Minorities and Women: Chartbook. American Public Health Association, Washington, DC, 1982.
505. Kunitz SJ, Levy JE. Navajos. In: A Harwood (Ed) Ethnicity and Medical Care, Harvard University Press, Cambridge, MA, 1981, pp 337-396.
506. Sievers ML, Fisher JR. Increasing Rate of Acute Myocardial InFarction in Southwestern American Indians. Arizona Medicine 36: 739-742, 1979.
507. U.S.DHEW. Selected Vital Statistics for Indian Health Service Areas and Service Units, 1972-1977. DHEW Publication (HSA) 79-1005.
508. Kumanyika SK, Savage DD. Ischemic Heart Disease Risk Factors Among American Indians and Alaska Natives. Paper commissioned By the DHHS Task Force on Black and Minority Health, 1984-85.
509. Fulmer HS, Roberts RW. Coronary Heart Disease Among the Navajo Indians. Annals of Internal Medicine 59:740-764, 1963.
510. Markides KS. Mortality Among Minority Populations: A Review of Recent Patterns and Trends. Public Health Reports 98(3): 252-260, 1983.
511. Gillum RF, Gillum BS, Smith N. Cardiovascular Risk Factors Among Urban American Indians: Blood Pressure, Serum Lipids, Smoking, Diabetes, Health Knowledge, and Behavior. American Heart Journal 107(4):765-776, 1984.
512. Arizona's Pima Indians Are an Apparent Exception to the Obesity - High Cholesterol - Heart Disease Pattern. The NIH Record DHHS, Volume 37(1), January 2, 1985.
513. Sievers ML. Historical Overview of Hypertension Among American Indians and Alaska Natives. Arizona Medicine 34: 739-742, 1979.
514. DeStefano F, Coulehan JL, Wiant MK. Blood Pressure Survey on the Navajo Reservation. American Journal of Epidemiology 109: 335-340, 1979.
515. Gillum RB, Prineas RJ, Palta M, Horibe H. Blood Pressure of Urban Native American School Children. Hypertension 2:744-749, 1980.
516. Sievers ML. Serum Cholesterol Levels in Southwestern American Indians. Journal of Chronic Diseases 21:107-115, 1968.
517. Ingelfinger JA, Bennett PH, Liebow IM, Miller M. Coronary Heart Disease in Pima Indians. Electrocardiographic Findings and PostMortem Evidence of Myocardial Infarction in a Population With a High Prevalence of Diabetes Mellitus. Diabetes 25:561-565, 1976.
518. Howard BV, Zech L, Davis M, Bennison LJ, Savage PJ, et al.

Studies of Very Low-density Lipoprotein Triglyceride Metabolism in an Obese Population with Low Plasma Lipids: Lack of Influence of Body Weight on Plasma Insulin. Journal of Lipid Research 21:1032-1041, 1980.
519. Howard BV, Davis MP, Pettit DJ, Knowler WC, Bennett PH. Plasma and Lipoprotein Cholesterol and Triglyceride Concentrations in the Pima Indians: Distributions Differing From Those of Caucasians. Circulation 68:714-724, 1983.
520. Garnick MB, Bennett PH, Langer T. Low-Density Lipoprotein Metabolism and Lipoprotein Cholesterol Content in Southwestern American Indians. Journal of Lipid Research 20:31-39, 1979.
521. Sievers ML, Fisher JR. Diseases of North American Indians. In: H.R. Rothschild (Ed.) Biocultural Aspects of Disease. Academic Press, New York, 1981, pp 191-252.
522. Brod MD, Thomas M. Alcoholism as a Mental Health Problem of Native Americans: A Review of the Literature. Archives of General Psychiatry 91:32-36, 1979.
523. Porter MR, Vienna TA, Kaplan GJ, Husch JR, Colyar AB. Drug Use In Anchorage, Alaska: A Survey of 15,634 Students in Grades 6 Through 12, 1971. Journal of the American Medical Association 223:657-664, 1973.
524. Taylor TW. The States and the Indian Citizens. U.S. Department of the Interior. Bureau of Indian Affairs, Washington, DC, 1972.```


[^0]:    * Small studies in the South revealed lower incidence of coronary $*$ * disease in Black than in white men. Among women, Blacks had * * similar or slightly higher rates than whites. Studies of hos- * * pital admissions for acute myocardial infarction indicate higher * * rates for whites than for Blacks. This may reflect higher Black *怠 rates of sudden death before hospital admission.
    

[^1]:    * 
    * Cardiovascular and cerebrovascular diseases in Hispanics result \%
    * from the interplay of many factors, some of them biomedical and *
    * others more dependent on socioeconomic and sociocultural context. $\%$
    * A reductionistic model of disease, that ignores social context, *
    * is inadequate for accurate assessment and successful treatment. *
    

[^2]:    From: Vital Statistics of the U.S., National Center for Health Statistics

[^3]:    From: "Coronary Heart Disease in Black Populations: Mortality and Morbidity", Gillum, R.F. [Table III]. American Heart Journal 104(4;1):844, 1982

[^4]:    $1_{\text {Elevated }}$ blood pressure (that is, a systolic measurement of at least 160 mm Hg or a diastolic measurement of at least 95 mm Hg ) or taking antihypertensive medication.
    ${ }^{2}$ Reported never told by physician that he or she had high blood pressure or hypertension.
    ${ }^{3}$ Subset of "On medication" group; those taking antihypertensive medication whose blood pressure was not elevated at the time of the examination.
    ${ }^{4}$ Age adjusted by direct method to the population at midpoint of the 1976-80 National Health and Nutrition
    ${ }_{5}{ }^{\text {Examination Survey. }}$
    From: Blood Pressure Levels and Hypertension In Persons Ages 6-74 Years, U.S. 1976-81. Advance Data No. 84, DHHS

[^5]:    From: "Stroke Report," Kuller, L. [Table 2]. Paper commissioned by

[^6]:    From: "Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol
    five years," Neaton, J.D. et al. [Table II]. Amertcan Heart Journal 108(3;2):762, 1984
    concentration, and diastolic blood pressure among black and white males followed up for

[^7]:    $\begin{array}{ll}\text { From: } & \text { Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol } \\ \text { concentration, and diastolic blood pressure among black and white males followed up for } \\ & \text { flve years," Neaton, J.D. et al. [Table III]. American Heart Journal 108(3;2):763, } 1984\end{array}$
    $\begin{array}{ll}\text { From: } & \text { Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol } \\ \text { concentration, and diastolic blood pressure among black and white males followed up for } \\ & \text { flve years," Neaton, J.D. et al. [Table III]. American Heart Journal 108(3;2):763, } 1984\end{array}$

[^8]:    From: "Stroke Report," Kuller, L. [Table 52]. Paper commssioned by the Task Force on Black and Minority Health, 1984-85

[^9]:    From: "Race, Education, and Five Year Mortality in HDFP Stratum I
    Referred Care Males. In F Gross and T Strasser (Eds)
    Mild Hypertension: Recent Advances, Raven, New York, 1983,

[^10]:    From: "A note on the biologic concept of race and its application in epidemiologic research", Cooper, R. [Table I]. American Heart Journal 108(3;2):720, 1984

[^11]:    a. Per Capita Income = Family Annual Income Divided by Number of People Sharing the Income.
    b. Weight $=$ Percent of Ideal Body Weight.
    c. Normotensive = Diastolic Blood Pressure (DBP) Less Than 90 mm Hg and Not On Antihypertensive Medication.
    d. Hypertensive $=$ DBP at Least 90 mm Hg and/or On Antihypertensive Medication.
    e. Controlled $=$ DBP Less Than 90 mm Hg and On Antihypertensive Medication.
    f. Mild = DBP 90 to 104 mm Hg .
    g. Moderate to Severe $=$ DBP at Least 105 mm Hg .

[^12]:    From: "Race, Education, and Five Year Mortality in HDFP Stratum I
    Referred Care Males. In $F$ Gross and $T$ Strasser (Eds)
    Mild Hypertension: Recent Advances, Raven, New York, 1983,

[^13]:    From: "Financial Cost as an Obstacle to Hypertension Therapy", Shulman NB, Martinez B, Brogan $D R$, Carr $A A$, Miles $C G$ (submitted for publication)

[^14]:    From: "The Incidence of Treated End Stage Renal Disease In the Eastern United States: 1973-1979", Sugimoto, T and Rosansky, S.J. [Figure 5]. American Journal of Public Health 74(1):16, 1984

[^15]:    From: "The Contribution of the Social Environment To Host Resistance", Casse1, J. [Figure 4]. American Journal of Epidemiology 104(2): 116, 1976

[^16]:    From: "Office Visits for Diseases of the Circulatory System", the National Ambulatory Medical Care Survey, United States, 19751976." DHEW Publication No. (PHS) 79-1791, January 1979

[^17]:    From: "Cardiovascular Diseases in Los Angeles County, 1978-1981, Frerichs, R.R. et al. American Heart Association. Greater Los Angeles Affiliate, Inc., 1983

[^18]:    From: "Cardiovascular Diseases in Los Angeles County, 1978-1981", Frerichs, R.R. et al. American Heart Association. Greater Los Angeles Affiliate, Inc., 1983

[^19]:    From: "Asian-white mortality differentials: Is there excess death?", Yu, E.S.H. et al. Paper commissioned by the Task Force on Black

[^20]:    *Percent values are not given when the bage of calculation (the excess deaths from all causes for a particular age, sex, and racial group) is equal to zero.
    From: Mortality Rates, Excess Deaths. National Center for Health Statistics' Death Certificate Data Tapes for 1979 , 1980 , and 1981. (Tables supplied by the DHHS Task Force on Black and Minority Health.)

[^21]:    *Percent values are not given when the base of calculation (the exceas deaths from all causes for a particular age, sex, and racial group) is equal to zero.
    From: Mortality Rates, Excess Deaths. National Center for Health Statiatics' Death Certificate Data Tapes for 1979, 1980, and 1981. (Tables supplied by the DHHS Task Force on Black and Minority Health.)

[^22]:    From: "Asian-white mortality differentials: Is there excess death?", Yu, E.S.H. et al. Paper commissioned by the Task Force on Black and Minority Health, 1984-85

[^23]:    From: "Asian-white mortality differentials: Is there excess death?", Yu, E.S.H. et al. Paper commissioned by the Task Force on Black and Minority Health, 1984-85

[^24]:    *As identified on the death certificate.
    +Percentage of total deaths in the specified race-ethnic group.

[^25]:    From: "Cardiovascular Diseases in Los Angeles County, 1978-1981," Frerichs, R.R. et al.

[^26]:    From: "Cardiovascular Diaeases in Los Angeles," Chapman, J. M. et al. Los Angeles, CA. American Heart

[^27]:    From: "Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: Incidence of stroke in Japan and Hawaii", Takeya, Y. and Popper, J.S.
    Stroke 15:15-23, 1984

